

THE BRITISH
JOURNAL OF SURGERY



THE BRITISH JOURNAL OF SURGERY

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THE BRITISH JOURNAL OF SURGERY

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, K.B.E., LONDON

X. A CASE OF ACUTE PERFORATIVE APPENDICITIS

As this number of the BRITISH JOURNAL OF SURGERY is issued to commemorate the good service Lord Moynihan has rendered it for more than twenty years, it may not be inappropriate to recall one of the earliest, if not the very earliest, of his publications. He read the case at a meeting of the Leeds and West Riding Medical Society in May, 1895, when he was aged 30, and was an Assistant Surgeon at the Leeds General Infirmary. It is entitled, "A Case of Acute Perforative Appendicitis followed by Septic Peritonitis; Abdominal Section: Recovery". The communication afterwards appeared in *The Lancet* (1896, ii, p. 1806), and is here slightly shortened. It runs:—

"Cases of recovery after abdominal section performed for acute septic peritonitis depending on disease of the vermiform appendix are sufficiently rare to warrant the publication of any that may occur. In the early part of last year I was fortunate enough to have such a case. The patient, aged 13, an exceptionally bright and intelligent boy, suffered two years ago from some illness of one week's duration, characterized by abdominal pain, constipation, and a general feeling of languor and sickness without actual vomiting. From this date until March 29th he was perfectly well, but on that day he had in the early morning a loose stool with some abdominal discomfort hardly amounting to pain. The pain continued to increase until on April 1st it had become serious and was accompanied by abdominal distension. Examination showed that the pulse was rapid and feeble, the temperature 100·6° F., the skin clammy and the abdomen uniformly distended and everywhere tender but without any localized resistance. An operation was decided upon, the general peritoneal cavity being first opened by an incision through the linea alba and then cleansed thoroughly, the cæcal region being afterwards exposed through a separate incision and dealing with whatever was found there.

"On opening the peritoneal cavity a large quantity of curdy looking, highly offensive pus made its escape. On introducing my finger into the abdomen I was able to make out that there was a moderate sized collection of pus in Douglas' pouch and I could feel an opening distinct though small which led to an abscess cavity in the neighbourhood of the appendix in the iliac fossa. I then thoroughly cleansed the whole peritoneal cavity, spending some time over it and taking pains

to ensure that no small collection of pus hemmed in by adherent coils of intestine, escaped notice. I then put a plug of iodoform gauze into the cæcal region through the opening I had found and incising the wall above Poupart's ligament I exposed the abscess cavity. Its lining was shreddy, irregular, and in places almost gangrenous and though I have no doubt that the appendix was the cause of the formation of the abscess I was unable to identify it unmistakably and therefore could not remove it. I cleansed the abscess as perfectly as I could by scraping



LORD MOYNIHAN, aged 30

gently, sponging firmly with iodoform gauze and flushing with boiled water. I then dusted the whole cavity with a little iodoform and employed a glass tube to drain the general peritoneal cavity and a stout indiarubber tube to drain the cæcal region.

"The patient rallied after the operation much better than we either hoped or expected. He remained in bed for nine weeks and though in danger for perhaps the first sixty hours, there was no doubt after that time that the ultimate issue of the case would be satisfactory. The boy is now quite recovered and as healthy and vigorous as ever.

"The class of case similar to the one I have just described is of course almost invariably fatal. During the whole of the time that I was house surgeon and resident surgical officer at the [Leeds] Infirmary there was not a single case of recovery from this condition and until the last two or three years not a single recovery had, so far as I know, been put on record and even up to the present time the successful cases in England number only units. But that recovery should be, and will eventually be, the rule instead of the exception I feel complete confidence. In pondering over the matter I have thought that it might be possible to formulate certain essentials on the strict observance of which I feel complete confidence. They are (1) Make an early diagnosis; (2) If at the end of say twenty-four hours the symptoms are not retrogressing operate at once; (3) If the case is first seen when the abscess has formed do not be content with merely opening and draining the cavity, but, if possible remove the appendix as well; (4) If the case is not seen until an early diagnosis of perforation has been made operate immediately, cleansing thoroughly but avoiding, if possible, lavage of the peritoneum. If these four rules for treatment be efficiently carried out I believe that a larger percentage of cases will recover than ever before and that every patient who has recovered will be exempt from any similar attacks in the future."

It should be remembered that Moynihan gave this advice in 1895, when inflammation of the appendix was just coming into notice. The number of operations at St. Bartholomew's Hospital in that year was 10, and 3 of the patients died. The general surgeons still feared the peritoneum, and there was a distinct cleavage of surgical opinion as to the treatment of a condition with which they were as yet unfamiliar. The older school advised waiting until the acute symptoms had passed away. They operated when there was an abscess or after a delay of three weeks. The younger generation advocated immediate operation, and to this school Moynihan belonged.

I am indebted to Lady Moynihan for the photograph, which she believes was taken in or about the year 1895.

LORD MOYNIHAN

A PERSONAL APPRECIATION

BY RUTHERFORD MORISON, NEWCASTLE-UPON-TYNE

IT is almost fifty years since I first met Moynihan. I was then in practice at Hartlepool, and had operated upon a patient in whom he was interested. He wrote asking if I could see him and tell him all about the case if he came through—and he did. I suspect, too, that he wanted to see what sort of fellow I was.

He was then tall, thin, and very pale, but so alert that he would now be described as a "real live wire"; but with complete control. Nothing escaped his notice, and he wanted to know more than I could tell him about everything. At lunch we were honoured, as I always was, by the company of an enormous castrated tom cat, and afterwards we walked to the hospital to visit some malaria patients under my care. On my eightieth birthday, two years ago, he wrote the kindest letter, and asked if I remembered the cat and the malaria. Of course I did, and I also recall that as we walked to the hospital he said he did not think the majority of surgeons knew enough anatomy—they should at least know the landmarks of the epiphyses and remember when their fusion with the shafts occurred.

I remember so clearly our conversation on this first meeting because at this time I was mentally starved, so far as my professional life was concerned. My colleagues in Hartlepool were old and had long given up any idea of inquiring into the why and wherefore of their work. The delight of meeting and talking with an enthusiast such as Moynihan can be readily imagined. We did not then, and have not since, always seen eye to eye on all the problems we tackled; but our differences of opinion have but added to the zest and enjoyment of our long friendship.

Hartlepool, when Moynihan visited it, was a very old seaport and fishing town between the Tees and the Tyne on the east coast, sleepy but holding its position chiefly as an importer of timber and exporter of coal.

The next time I saw Moynihan was at Leeds Infirmary, where I had gone to see Mayo Robson operate. Moynihan was Resident Surgical Officer, and his chief interest at that time was in fractures. From this time I noticed with interest Moynihan's steady and inevitable rise to the highest attainable positions, gained by the sheer force of such irresistible ability as could not be ignored.

In the following years I frequently met him. About fifteen years ago I visited Leeds and saw him perform two operations. The first patient was a thin fragile old lady with a long history of stomach trouble, pain, frequent vomiting, and a lump at her pylorus, the result of chronic ulceration of the lesser curvature involving the pylorus. Half of her stomach was excised. The second patient was a fat middle-aged woman sent in as an abdominal emergency. She had been suddenly seized by overwhelming pain and vomiting, and had developed a high temperature. Her swollen gall-bladder could be felt, and an acutely inflamed, tense, and nearly gangrenous gall-bladder was excised—a typical example of those cases where a stone blocks the neck of the gall-bladder when it has just started to empty itself and the

septic bile cannot escape. Acute infection and tension complete the destruction.

Both of these operations thrilled me, because they showed such perfect art. What is the secret of such success? Surely that the surgeon has learned to make his operation so easy that any hitch was incredible. We have all heard how *easy* golf is to Bobby Jones, and, for the same reason, I do not doubt it.

Though Lord Moynihan's skilful and beautiful operations will not soon be forgotten, my feeling is that the future will attach more importance to his founding of the Moynihan Chirurgical Club. He has drawn together a group of the rising younger surgeons to educate themselves by meeting twice a year: once to spend a week together in hospitals abroad, and the second time in a daily visit to a hospital in which one of them works. It is difficult to over-estimate the value of this experience, not only to the surgeons and their patients, but to the whole community.

(R. M.)

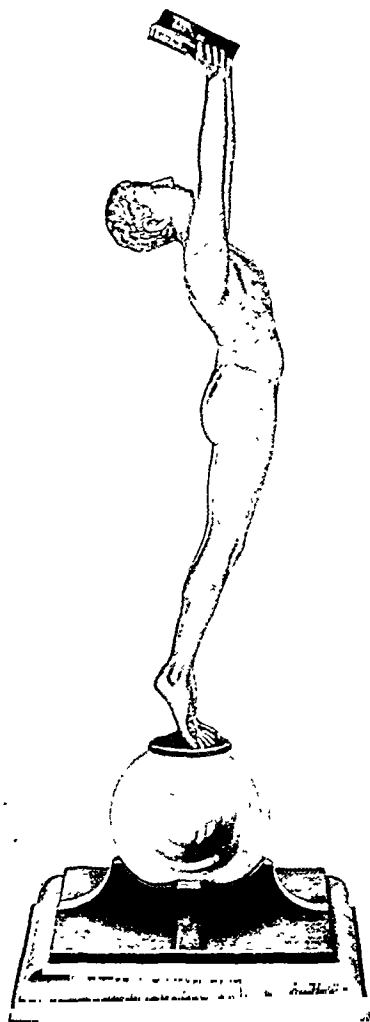
It was in the years 1912-13 that the two projects of the Association of Surgeons and the BRITISH JOURNAL OF SURGERY began to take shape. The membership of the Chirurgical Club was limited to provincial surgeons. Its *raison d'être* was to bring closer association between provincial surgeons. This having been accomplished, it then remained to provide for a closer union between provincial and metropolitan surgeons and to found a journal representative and expressive of British surgery. Sir Rickman Godlee at that time was the President of the Royal College of Surgeons. It was in the drawing-room of his house that Moynihan first mooted his proposal for founding the Association of Surgeons to an audience of leading London surgeons. It was accepted, though with some doubt and criticism. But the matter had not proceeded beyond the stage of preliminary discussion when the war postponed any decisive step, so that it was not until 1919 that the Association was founded with Sir John Bland-Sutton as its first President.

Fortunately the BRITISH JOURNAL OF SURGERY had been planned and started before the war began, so that it first appeared in April, 1913, with Moynihan as the Chairman of the Editorial Committee. That Committee has always been representative of all the teaching schools of England, India, and the Dominions. It was faced with a momentous decision in August, 1914, when the proposal was seriously made to postpone further publication until the war was over. It was then thought that this would only be for a period of at most six months! But it was decided to "carry on" and to seize the opportunity of recording the history of British surgery in the war. This was a fortunate decision, because it brought us into close touch with Belgian, French, and American surgeons. Moynihan was then the Chairman of the Consulting Surgeons attached to the British Army, and his wise guidance of the BRITISH JOURNAL OF SURGERY helped to establish the new venture in a position of assured success.

During the six years of his Presidency of the Royal College of Surgeons, Moynihan lost no opportunity of encouraging the promotion of surgical research, both at the laboratories of the College and later on at the Buckston Browne Farm.

The JOURNAL has been born, it has survived the dangers of infancy and adolescence, and grown to adult age during the more than twenty-one years that Moynihan has presided over its destinies. During that time he never missed a meeting of the Editorial Committee, and these meetings have always been as happy under his chairmanship as they have been fruitful of good results.

It is in grateful recognition of the great part which Lord Moynihan has played in the founding and editing of this JOURNAL and in the promotion of surgical research that a testimonial is presented to him by the Editorial Committee and by the subscribers to the BRITISH JOURNAL OF SURGERY. This takes the form of a



silver statuette of symbolic character designed and made by Mr. Omar Ramsden; of the gift of a thousand guineas to the Council of the Royal College of Surgeons for the promotion of research; and of a tablet placed on the walls of the College, perpetuating our sense of indebtedness to a great surgeon and a great leader.

TOTAL REMOVAL OF A LUNG

By R. MILNES WALKER

HONORARY SURGEON, ROYAL HOSPITAL, WOLVERHAMPTON

THE subject of total removal of a lung is one which has had increasing prominence in the literature during the last few years. Though the operation was first attempted as long ago as 1910, no successful case was reported until 1931. Up to the present there have been reports of 34 cases in the literature and references to a further 10. Of 36 cases where the diagnosis is given, in 19 the operation was performed for a tumour of the lung, and in the remaining 17 for inflammatory conditions. Of the total of 44 cases, 24 have survived the operation, giving a case mortality of a little under 50 per cent. These results are steadily improving, and the figures for the last year or two are considerably better than this.

The problems connected with this extensive operation have recently been admirably summarized in a paper by Overholt,¹ who gives a careful description of a series of 8 personal cases. There is no doubt that in the present limited experience of the operation no definite rules can be laid down. The present writer will, therefore, enumerate the main problems connected with this operation, particularly as performed for inflammatory conditions in the lung, and give a report on a personal case together with a table of the cases so far recorded. It was felt that such a table might throw light on some of the more important dangers of the operation, with particular reference to the fatal cases where the cause of death is stated.

There are two separate groups of cases in which this operation of removal of a whole lung has been performed: firstly, bronchiectasis and multiple abscesses in the lung, and, secondly, new growths. In cases of bronchiectasis, it is necessary to ascertain by means of a bronchogram that the disease is entirely limited to one side, but in those cases in which both upper and lower lobes are involved, and which have not yielded to less severe measures, the operation certainly has a field of usefulness; whether it will displace a thoracoplasty for such cases remains to be seen after further experience, but most thoracic surgeons seem to be agreed that the results of the latter operation in this disease are disappointing. Regarding malignant growths of the lung, the increasing use of the bronchoscope is certainly leading to earlier diagnosis, and where there is a possibility of radical extirpation of the disease by means of pneumonectomy, the attempt is certainly worth making. How permanent as regards the question of recurrence the results will be, can only be ascertained after a much longer experience, but the supervention of both local recurrence and cerebral metastases has already been recorded in cases where it had been hoped that the disease had been entirely eradicated by complete removal of the lung.

The problem is somewhat different in the inflammatory cases from what it is where a new growth without subsequent inflammation exists. No definite rule can be laid down regarding the choice of a single-stage or a multiple-stage operation;

in most of the early cases a multiple-stage procedure was employed, but the recent tendency is towards completing the whole operation at a single sitting. The presence of only one fatality from shock in the whole series (in a tuberculous case with very extensive adhesions) leads one to consider that with adequate means for intravenous infusion or blood transfusion, patients stand this extensive operation remarkably well, and the need for performing the operation in multiple stages will be confined to those cases only in which there are very dense adhesions.

In some respects the problems are similar to those confronted in cases of lobectomy, but there is the important difference that in lobectomy the remaining portion of the lung still occupies the pleural cavity, and steps have to be taken to maintain its expansion and to encourage it to fill the whole cavity on that side, whereas in pneumonectomy there is the need for the obliteration of this cavity.

One of the first considerations is the necessity for preliminary pneumothorax; here the problem closely resembles that in lobectomy, but the writer feels that where a pneumothorax is possible it should be carried out ten to twelve days before the major operation. In cases of bronchiectasis, however, such as the one now recorded, the adhesions between the lung and chest wall frequently make this impossible. In the inflammatory cases the freeing of these adhesions may occupy the greater part of the operation; in these cases also the structures of the hilum may be matted together by fibrous tissue, so that their dissection and individual ligature is impossible. Where the operation is undertaken for a new growth, however, there are not likely to be adhesions, unless an inflammatory process has supervened, and the approach to the hilum is not nearly so difficult; it is in these cases that the anterior incision has been found to give good access to the hilum, whereas in the inflammatory cases the postero-lateral route appears to be more satisfactory.

As regards the anæsthetic, the writer has used nitrous oxide and oxygen for both lobectomy and total pneumonectomy. Postural drainage is carried out immediately before the patient is taken to the operating theatre, in order to drain the lung as completely as possible. Where there is little or no sputum there appears to be no need for intratracheal intubation, and in the present case the anæsthetic proved quite satisfactory with a closed mask and positive pressure, without intubation. The lateral movements of the mediastinum, however, were somewhat alarming during the course of the operation, but this was steadied to some extent by the assistant by means of a clamp on the diseased lung during the early part of the operation, and by fixation of the tourniquet on the pedicle, after this was applied. The postero-lateral approach was used and gave a very satisfactory exposure of the hilum, a long incision being made in the 6th intercostal space. The residual space was drained by means of closed drainage and a water seal, which seems to be a necessity where there has been risk of infection of the pleural cavity, though a number of cases are recorded, particularly where the operation has been performed for neoplasms, in which primary closure of the chest has been effected, and aspiration of serum performed only when necessary afterwards.

The necessity for a subsequent thoracoplasty appears to depend on a number of factors, but the present case shows that it is by no means necessary in all instances, and, particularly in children, the chest wall and mediastinum are capable of adapting themselves to the changed conditions without either a phrenicectomy or a thoracoplasty.

The means of dealing with the hilum is a matter which is by no means settled, but is perhaps the most fundamental step of the operation. All grades of technique are being practised by different observers. First of all there is mass ligature of the hilum, allowing the lung to separate naturally, which is apt to happen after about fourteen days. This method, which was practised by Nissen in the first case of successful pneumonectomy, and has since been followed by Mason, has the advantage that it reduces the time of the operation and does away with the danger of primary hæmorrhage from the pulmonary vessels. It entails, however, the necessity of leaving a widely open wound, containing the necrotic lung, with infection of the pleural cavity, and the certainty of a bronchial fistula when the lung separates. Subsequent anæsthetics may be necessary for dressing the wound, and in one case the lung, which had failed to separate after thirty days, had to be removed with the cautery. Then there is the mass ligature of the hilum with primary amputation of the lung; this is also very prone to give rise to a bronchial fistula and infection of the residual space. The next method is the application of a tourniquet and suture of the pedicle with mattress sutures; this has been practised successfully by Roberts and Tudor Edwards, and was performed in the author's case without any subsequent bronchial fistula. A Shenstone-Janes tourniquet of silk was used, and the lung amputated by diathermy $\frac{1}{4}$ in. distal to the tourniquet, the short thickened pedicle making it impossible to apply a second tourniquet on the pulmonary side; any escape of pus from the bronchi was carefully prevented by means of a swab. Five mattress sutures of catgut were then inserted right through the pedicle, and the tourniquet removed. A few further catgut stitches were put in, approximating as far as possible the mediastinal pleura in front of and behind the pedicle, though there was insufficient free pleura to cover the stump completely; the bronchial stump had retracted to some extent but was not entirely buried. Judging from the writer's and others' experience of lobectomies, primary closure of the bronchial stump may be obtained by this method in the majority of cases. The fourth method of dealing with the hilum is the dissection of its individual structures, and their separate ligature. The risk in this is injury to the pulmonary artery, and a number of cases are recorded where fatal hæmorrhage from this artery has occurred while this method was being practised. Its advantage in cases of malignant disease is obvious in that the bronchus may be ligatured more proximally and glands can be dissected out from the hilum, but it is particularly dangerous in cases of inflammatory disease of the lung, where there is apt to be considerable fibrosis among the structures of the hilum, and in these cases it appears to have no advantages over the tourniquet and suture method.

CASE REPORT

Male, aged 7 years, was first admitted to the Royal Hospital, Wolverhampton, in June, 1929, at the age of 11 months. There was a history of cough for three months, and difficulty with breathing for six weeks. A diagnosis of bronchitis was made, and after a fortnight he returned home. Four months later he was re-admitted with signs suggesting bronchopneumonia in the left lower lobe; this subsided, but the cough persisted, and he continued to attend the hospital, being an in-patient again in November, 1930, when he is reported to have had consolidation of the left lower lobe; on being re-admitted for the fourth time four months later, the consolidation was again noted at the base of the left lung, but there was no fever. In December, 1930, both antra, which contained mucopus, were washed out, and the following month the tonsils and adenoids were removed. He was not in hospital again for

four years, but during that period the cough continued unabated, and he attended the Out-patient Department spasmodically.

In February, 1935, he came into hospital, having had measles three weeks previously, followed by pains in the chest; the cough was as bad as ever; he was bringing up from 1 to 4 oz. of offensive sputum daily, clubbing of the fingers was present, and there was dullness and impaired entry over the whole of the left side of the chest. On aspiration of the left base, 5 c.c. of purulent fluid were withdrawn, and this, on culture, yielded pneumococci. An X-ray examination showed the left chest to be completely opaque with poor movement; the right lung field was clear, and the heart was displaced a little to the left. During the following two months his condition remained stationary; he had no fever most of the time, but every two or three weeks he had a febrile attack lasting two or three days, the temperature rising to about 101° . In April he had one of these attacks which was much more severe than his previous ones; the temperature rose to 103° , the pulse to 150, and respirations to 56; some crepitations were present at the right base, but the physical signs on the left side remained

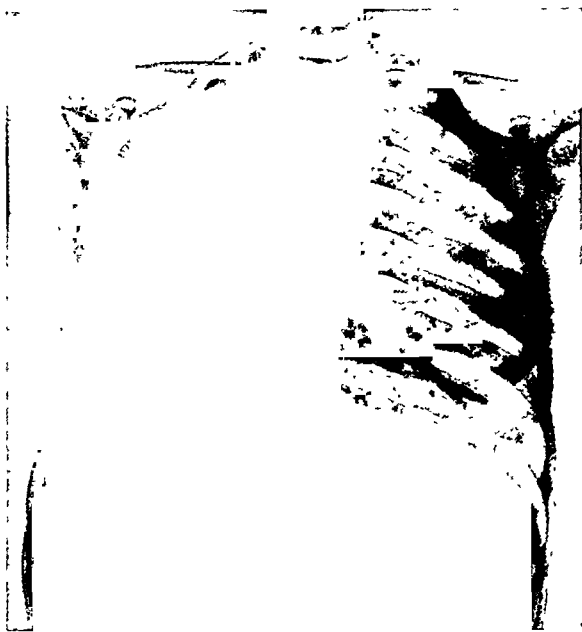


FIG. 1.—X-ray of chest before operation.

unchanged. A further small quantity of pus was aspirated from the left side. On this occasion the fever took two weeks to subside; he then developed an alveolar abscess for which an upper molar tooth was extracted.

A lipiodol injection in May showed bronchiectatic cavities in both upper and lower lobes on the left side, but the right lung was clear. Postural drainage did not improve his condition, and it was decided to attempt a pneumonectomy on the first favourable occasion after he had remained free from a febrile attack for some weeks. Attempts at a preliminary artificial pneumothorax were unsuccessful. *Figs. 1-3* show the radiographic appearances before and after operation.

OPERATION.—The operation was performed at the Royal Hospital, Wolverhampton, on Sept. 3, 1935. The anæsthetic, administered by Dr. J. H. Richmond, was nitrous oxide and oxygen, without intubation of the trachea, positive pressure being maintained with a closed mask. A long incision was made along the 6th intercostal space, and a small portion of the 6th rib was removed near its angle. The lung was found to be completely adherent, but the adhesions could be separated with the fingers without much difficulty, except at the apex and



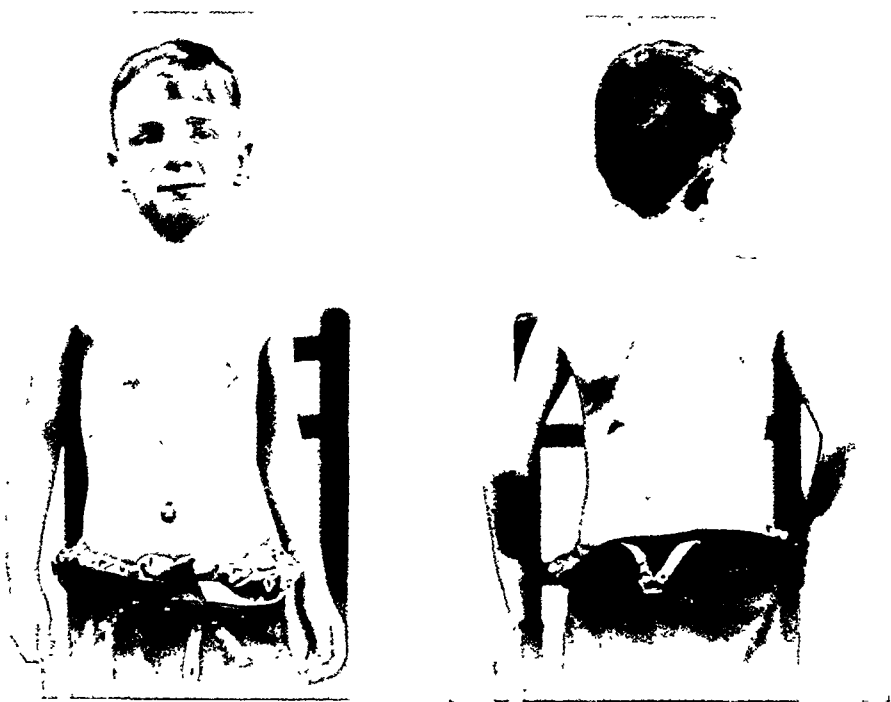
FIG 2—Bronchogram before operation



FIG 3—X-ray of chest one month after operation

the diaphragm, where they were divided by diathermy. The lung was then freed on all sides down to the hilum, as close to the mediastinum as possible, and the lung amputated by diathermy $\frac{1}{2}$ in. distal to the tourniquet. The pedicle was then secured with mattress sutures of catgut, and the tourniquet removed; a few further stitches were then placed to approximate the pleural margins as far as possible, but the stump could not be completely buried. Intercostal drainage was provided by means of a self-retaining catheter through a stab in the 9th intercostal space, and the wound closed with catgut sutures, leaving a small rubber drain into the subcutaneous tissues. The catheter was connected to a water seal. The pulse-rate, which had been 120 at the beginning of the operation, had risen to 140, but there was remarkably little shock, and at no time was it necessary to administer fluids except by mouth.

SUBSEQUENT PROGRESS.—On the fourth day the intercostal drain came out, and no further drainage occurred from the stab-wound; there was a little pus from the main incision, but



FIGS. 4, 5.—The patient three months after operation. The vertical line drawn just outside the left nipple line marks the right border of cardiac dullness.

this appeared to originate in the subcutaneous tissues. Five days after operation the right border of cardiac dullness was $\frac{1}{2}$ in. inside the left nipple line; on the tenth day he coughed up 4 oz. of thick ropy pus, evidently an accumulation round the stump which had discharged itself into the bronchus; apart from this, the sudden cessation of his sputum following the operation was most marked. Two weeks after operation his wound was healed, and he left hospital on Oct. 10, thirty-seven days after operation. He continued to make further improvement, and eleven weeks after his operation was able to make the journey of 250 miles to London and back in the day, to be presented at a meeting of the Royal Society of Medicine.² (Figs. 4, 5).

At the present time, six months after the operation, he is in good health; he can run about and play with other children without getting breathless, though he has an occasional dry cough, but never brings up any sputum; the mediastinum is displaced to the left, the

right border of cardiac dullness being outside the left nipple line ; the heart appears to have become rotated backwards as well as to the left, for the heart-sounds are very loud over the back of the left chest, and cardiac pulsation can be felt in the 6th and 7th intercostal spaces in the scapular line ; there is slight dropping of the shoulder and flattening of the ribs on that side, but no scoliosis. His weight since the operation has increased from 39 to 45 lb.

PATHOLOGY.—The specimen of the excised lung shows that it is covered with thickened pleura, to which are attached a number of filmy adhesions ; a little adherent fat has come away with the lung at the apex, and the interlobar fissure is quite obliterated ; the inner aspect shows that the hilum has been divided at the point where the left bronchus divides into its two main branches ; the cut section shows the whole lung to be consolidated, with much fibrosis, especially in the lower lobe, and all the bronchi are moderately dilated. (*Fig. 6.*)



FIG. 6.—Vertical section of the excised lung.

A study of *Tables I and II* summarizing the cases of total pneumonectomy brings out a number of interesting points. There are references to 44 cases, 17 for inflammatory conditions, 19 for new growths, and 8 in which the diagnosis is not given (Churchill,²² 8 cases with 4 deaths). Of the total of 44, there have been 20 deaths, a mortality of 45 per cent, but in the 17 inflammatory cases the mortality is 29 per cent, and 59 per cent in the 19 cases of growths. The high mortality in the latter group appears to have a close connection with the more advanced age of the patients, for in those cases where the age is stated, there were 3 deaths among 16 cases under thirty (18·7 per cent), and 6 deaths among 12 cases over this age (50 per cent). Of 14 cases where the cause of death is stated, this was primary or reactionary hæmorrhage (3), secondary hæmorrhage (1), shock (1), mediastinal infection (1), pneumonia or œdema of the remaining lung (3), septicæmia (1), and other causes (4). It will be noted that there has been no death among the last 10 cases of bronchiectasis or pulmonary abscesses recorded. (*See Table III.*)

Table II.—TOTAL PNEUMONECTOMY FOR NEW GROWTHS, *continued*

AUTHORITY	DIAGNOSIS	AGE AND SEX	DATE OF OPERATION	ANÆSTHETIC	TREATMENT OF HILUM	RESULT
Reinhoff ¹⁷	.. Carcinoma, L. primary bronchus	42 F.	3/11/33	Avertin and nitrous oxide	Structures ligated separately	Recovery
Freedlander ¹⁹	.. Carcinoma, L. main bronchus	—	4/11/33	—	—	Died following subsequent thoracoplasty
Alexander ⁹	.. Carcinoma, L. bronchus ..	—	6/11/33	—	—	Died; cardiac failure, 30th day
Overholt ¹	.. Carcinoma, L. lower bronchus	33 F.	2/ 5/34	Ethylene ..	Suture ..	Recovery. Well 1 year later
Overholt ¹	.. Carcinoma, L. lung ..	43 M.	7/ 7/34	Cyclopropane	Suture ..	Died; 48 hours later; hæmorrhage from pulmonary artery invaded by growth
Overholt ¹	.. Carcinoma, L. main bronchus	37 M.	13/11/34	Cyclopropane and ethylene	Suture ..	Died; hæmorrhage from retracted pulmonary artery during operation
Overholt ¹	.. Carcinoma, R. lung ..	50 M.	8/12/34	Cyclopropane	Suture ..	Died; 6th day; pneumonia
Overholt ¹	.. Carcinoma, L. upper bronchus	59 F.	8/ 4/35	Cyclopropane	Structures ligated separately	Recovery
Graham ²⁰	.. Carcinoma	—	—	—	—	Died; 3½ weeks later, aspiration of fluid from bronchial fistula
Haight ²¹	.. Carcinoma	—	—	—	—	Recovery

Table III.—MORTALITY IN RELATION TO AGE
(28 CASES)

AGE	RECOVERED	DIED
Years		
0-9	5	2
10-19	6	1
20-29	2	0
30-39	3	3
40-49	2	2
50-59	1	1

It is yet far too early to assess the full value of this operation, and no prognosis can be given regarding the expectation of life with only one lung. In the case here recorded, however, the child's parents already feel that the operation has been well worth while, for both they and the patient have good nights' sleep instead of being disturbed, as they have been for five years, by his incessant coughing.

REFERENCES

- ¹ OVERHOLT, R. H., *Jour. of Thoracic Surg.*, 1935, v, 54.
- ² WALKER, R. M., *Proc. Roy. Soc. Med.*, 1936, xxix, 212.
- ³ MEYER, W., *Arch. of Surg.*, 1923, vi, 361.
- ⁴ LILIENTHAL, H., *Ann. of Surg.*, 1922, lxxv, 257.
- ⁵ NISSEN, R., *Zentralb. f. Chir.*, 1931, lviii, 3003.
- ⁶ WINDSBERG, E., *Jour. of Thoracic Surg.*, 1934, iv, 231.
- ⁷ ARCHIBALD, E., *Ann. of Surg.*, 1934, c, 796.
- ⁸ HAIGHT C., *Surg. Gynecol. and Obst.*, 1934, lviii, 768.
- ⁹ ALEXANDER, J. Quoted by Haight⁸.
- ¹⁰ MASON G. A., *Brit. Med. Jour.*, 1935, i, 299.
- ¹¹ GOWER, F. J. S., and EDWARDS, A. TUDOR, *Proc. Roy. Soc. Med.*, 1936, xxix, 221.
- ¹² ROBERTS, J. E. H., *Ibid.*, 220.
- ¹³ KUMMELL, H., *Verhandl. d. deut. Gesellsch. f. Chir.*, 1911, xl, 147.
- ¹⁴ IVANISSEVICH, O., and FERRARI, R.C., *Bol. y. Trab. de la Soc. de Cir. de Buenos Aires*, 1933, xvii. Quoted by Haight.⁸
- ¹⁵ LILIENTHAL, H., *Jour. of Thoracic Surg.*, 1933, ii, 600.
- ¹⁶ GRAHAM, E. A., and SINGER, J. J., *Jour. Amer. Med. Assoc.*, 1933, ci, 1371.
- ¹⁷ REINHOF, W. F., *Bull. Johns Hopkins Hosp.*, 1933, liii, 390.
- ¹⁸ OVERHOLT, R. H., *Jour. of Thoracic Surg.*, 1934, iv, 196.
- ¹⁹ FREEDLANDER, S. O. Quoted by Haight.⁸
- ²⁰ GRAHAM, E. A., *Jour. of Thoracic Surg.*, 1935, v, 78. Discussion on Overholt's paper.
- ²¹ HAIGHT, C., *Ibid.*, v, 79.
- ²² CHURCHILL, E. D., *Ibid.*, 80.

HYDATID DISEASE OF THE KIDNEY

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EVEN in countries where hydatid disease is common the kidney is affected in only a small percentage of patients. It is proposed in this article to report four cases together with a description of the renal echinococcosis in general and a review of the literature, more particularly that of the last ten years. For a systematic exposition of the subject of hydatids the reader is referred to the works of authorities, of whom Duvé, of Rouen, and Dew, of Sydney, occupy a foremost place. A brief sketch of the nature of the parasite and its mode of access to the kidney is, however, desirable before dealing more fully with the pathological manifestations in the urinary tract.

THE ECHINOCOCCUS IN GENERAL AND ITS MODE OF ACCESS TO THE KIDNEY

*Tænia echinococcus** or *Echinococcus granulosus* is one of the smallest of the Cestodes. Midget among its fellows of the tapeworm tribe, it gains secure refuge from the stress of the current and peristaltic motion of the canine intestine by lodging between the villi like a ship in dock. Minute as a fly's leg, only the base of its terminal proglottid presents to the bowel lumen. Hundreds of white spots amidst the dull red of the mucosa indicate its presence. Ceaselessly it devotes itself to the task of reproduction. Self-impregnated, the proglottid swells as its ovary fills to bursting point. Like a loaded vessel it casts off into the stream, the vacated berth being taken by the next of the series. Thousands of these ripe proglottides, each with its hundreds of ova, pass to the outer world to infest the pastures and pass into the stomachs of the grazing herbivora of the neighbourhood. Other eggs become attached to the hair and muzzle of the dog, ready to be transferred to the fondling hand and thence to the intestine of the farmer's child. Inspiration of the eggs as a method of infection was suggested by Dougan Bird in 1877, but is now considered exceptional, though Duvé has produced the disease experimentally by this route. In the slaughter-houses of Split, in Dalmatia, 85 to 100 per cent of the infected sheep—and nearly all are infected—have cysts only in the lungs (Račić). We are really as yet uncertain on the question of respiratory infection and, indeed, ignorant of the method by which the onchosphere attaches itself to the wall of the bowel and penetrates it to gain the circulatory system and the tissues. Dew observed the *fait accompli*, embryos in the portal veins of swine a few hours after

* In addition to *Echinococcus granulosus*, the common form, other species have been described—*E. oligarthus* in the jaguar (Diesing), *E. minimus* in the Macedonian wolf (Cameron), and *E. longimanubrius* in the Cape hunting-dog (*Lycan pictus*). No cystic forms of the last three have been identified except perhaps of *E. oligarthus* in the agouti or American guinea-pig. (T. W. M. Cameron, "Some Modern Biological Conceptions of Hydatid", *Proc. Roy. Soc. Med.*, 1926-7, xx, Pts. 1 and 2, 273).

ingestion. Having reached the blood-stream, the little animal is small enough to pass through the capillary system of the liver should it choose one of the wider capillary anastomoses. Its projecting hooklets may hold it up like an oxalate calculus in a ureter, or it may have a selective preference for the liver, for it stays there in the vast proportion of cases. A fair number, however, win through the right heart to the lung, though K. D. Fairley, impressed with the frequency of lower right lobe pulmonary cysts in man, revived the direct migration theory of Küster. Should the hexacanth pass through the lung filter, it reaches the left heart and has then all the organs and tissues of the body open to its invasion. The kidney is a favoured site, but cysts in it form a meagre 0·5 to 4 per cent of the whole in the various published series of cases.

Other routes to the kidney have been contemplated—by the thoracic duct and left subclavian vein, missing the liver; by the venous plexus of Retzius (Nicaise); and by direct migration from the duodenum or colon (Küster). The not infrequent occurrence of one cyst only in the body, and that in the kidney, instead of a series of liver and lung cysts strewn along the route of migration, gives ground for consideration of other than the usually accepted route; or, perhaps, there is a selective preference for one organ or the other—certain groups finding a liver or lung environment uncongenial. In the ultimate site of lodgement the embryo sheds its hooklets, and proliferates, secreting its fluid internally and its hyaline laminated membrane externally, and becomes the familiar hydatid cyst.

Eggs must often be swallowed in clumps, and at more than one time by those exposed—a charge of small shot, not a bullet. It follows that many hexacanthus must perish—swept out of the bowel or destroyed in the tissues—for one that survives. One cyst developed may act as a preventive for future infection in virtue of produced antibodies.

The embryo having reached the kidney, the amazing process of the second stage of reproduction begins. In man it is the last. The final stage of evolution depends on the consumption by the definitive host of the intermediate one. The cycle is between carnivora and herbivora. In the former the worm is in the intestine to be discharged, in the latter in the tissues to be eaten. Sheep and dogs provide the necessary field. Man is a side line. More fastidious than its big brother *Tænia solium*, which may be found in both phases in man, the adult echinococcus is never a parasite of the latter. Scolices swallowed or ruptured into his intestine pass out without further development.

The reproductive urge, however, goes on unabated. The whole system of quivering opalescent mother cyst, brood capsules, sand, pearl, or ruby daughter cysts, third generations, and separated scolices develops in the human subject, as in the animal. The potency of propagation staggers the imagination. Thousands of worms in one dog each producing a series of ripe proglottides in endless continuity; each proglottid with its hundreds of eggs; each egg able to produce an embryo and form a cyst; every cyst with its multitude of brood capsules and scolices (Dévé found 400,000 of the latter in a single cubic centimetre of hydatid sand); each scolex able to produce yet another cyst in the same host, with a geometrical progression of numbers, *ad infinitum*.

The scolex has indeed two lines of development open to it according to circumstances: in the tissues of the intermediate host, another cyst; in the intestine of the dog, a full-sized worm

FREQUENCY AND DISTRIBUTION OF RENAL HYDATID CYSTS

Nicaise, writing in 1914, had collected 474 cases of renal hydatid—377 from the literature and 97 received on invitation from doctors in many countries. Between that date and 1925, collective studies were published by Kretschmer, Russell and Kilbane, and others. Inclusive of a few pararenal cases noteworthy for their intimate relation to the kidney, the reports of the last ten years (1925-35) are given in the following table. It was compiled as the result of a fairly thorough search and may be considered as reasonably exhaustive :—

COUNTRY	No. OF ARTICLES PUBLISHED	No. OF CASES
Europe—		
Great Britain	4	4
France	6	9
Germany	4	3
Spain	3	3
Italy	12	24
Austria	1	3
Hungary	3	3
Holland	1	1
Czechoslovakia	1	1
Bulgaria	1	1
Rumania	2	2
Jugoslavia	2	10
Greece	1	46
Ukraine (U.S.S.R.)	5	5
U.S.S.R. (excluding Ukraine)	4	69
	— 50	— 184
Asia—		
Iraq	1	2
Siberia (U.S.S.R.)	2	2
Trans-Caucasia (U.S.S.R.)	1	1
Central Asia (U.S.S.R.)	1	6
	— 5	— 11
North America—		
United States	11	13
	— 11	— 13
South America—		
Argentina	13	17
Uruguay	3	3
Chile	1	1
	— 17	— 21
Africa—		
French North Africa (Tunisia and Algeria)	2	37
	— 2	— 37
Australia	4	20
	— 4	— 20
New Zealand	1	1
	— 1	— 1
Total ..	90	287

In this table the widespread distribution is indicated, but not the actual incidence. Iceland, once the most infested country, is not represented, though a great deal has been added to its hydatid history since the classic work of Finsen (1867). In richly journalized North America few of the scanty store of cases have escaped publication. Infested Arabia produced only two cases. China, Japan, and most of the smaller republics of America provide no reports. Taken as a whole, most cases no doubt remain unpublished. The interested student is especially commended to the collected series in the list: Gall (Russia, 66 cases); Toole (Greece, 46 renal and 10 pararenal); Goinard (French North Africa, 36 cases); and to the authors with most personal cases: Craig and Lee-Brown (16), Vila (8), Videnski (6), Oulié (4), Blum (4), and Račić (10).

A few articles in this decennium appeared without cases. They will be indicated in the appropriate place.

The world's literature contains a few reports of hydatid disease of the kidney every year. The list of 287, after subtracting collected cases, some of which were doubtless previously published, gives at least 161 new cases, making with the Nicaise series, and the twelve years subsequent, a total of approximately 800 cases, so that the features of this rare complaint are completely available for study.

The incidence of the disease fluctuates in a given country with the number of sheep, the living conditions, level of culture, and the efficacy of preventive measures. It is decreasing in Iceland and France, increasing in Siberia, the Argentine (Greenway), Greece (Toole), and Dalmatia (Račić). In England, though rare in man, it is said to be prevalent among sheep (Brailsford). In New Zealand it is keeping pace with the increase of population (Barnett).

NOMENCLATURE

The general build of an hydatid cyst is the same in all soft organs. The nomenclature has been somewhat confused, but the terms as here defined will be used throughout the article.

Pericyst.—This is the adventitia or boundary wall formed by the fibrocellular reaction of the tissues of the host.

Cyst.—The cyst is the cavity bounded by the pericyst with its contents.

Ectocyst.—The ectocyst is the limiting envelope of the parasite proper. The term will be used to embrace both the hyaline laminated membrane and the germinal layer within it. Considered separately the latter is sometimes known as the endocyst.

Pericystic Cavity.—This is a coined term to denote the potential and sometimes actual space existing between the pericyst and the ectocyst.

Endocystic Cavity.—This expression will be used to indicate the space bounded by the ectocyst. Its contents are hydatid fluid with brood capsules, free scolices and hooklets, and sometimes daughter cysts and their contents.

RELATION OF CYSTS TO THE RENAL PELVIS

There are three classes of renal cyst according to the relation maintained to the kidney pelvis—namely, open, closed, and pseudo-closed. In the first both pericystic and endocystic cavity communicate with the pelvis, in the second neither, and in the third only the pericystic cavity, the ectocyst remaining unruptured.

Characteristic symptoms are found corresponding roughly to this classification. In the open cysts—renal colic and the passage of hydatid elements in the urine; in the closed the symptoms are mostly due to pressure; in the third the typical manifestations are intermittent attacks of localized loin pain due to calix colic, and perhaps urinary symptoms such as hæmaturia or frequency, but no hydatid material is passed.

CASE REPORTS

Of the four cases only the first lent itself to a systematic study. In the second a destroyed kidney was removed twenty-three years after the original operation for hydatid disease. In the third the patient refused investigation. In the fourth the diagnosis was confirmed at autopsy. The second and third cases are only given in brief.

Case 1.—Female, aged 38. Single.

HISTORY.—The patient had lived in sheep country all her life. She suffered from left renal colic at the age of 18. Passed some membranous material unidentified. No further trouble. At the age of 21 and again at 24, laparotomy was performed for vague abdominal pains. A small corpus luteum cyst was removed at the first operation, a normal appendix at the second. Thereafter the patient was perfectly well till seven days before consultation. One evening

she experienced agonizing radiating pain in the left loin. This passed off and next morning she had retention of urine. After being relieved by catheter, she passed a "white grape-like body". Her medical attendant found this to be hydatid membrane and referred her with the diagnosis of hydatid cyst of the left kidney.

ON EXAMINATION (July 12, 1935).

—The patient's general condition was good and all organs were normal. Neither kidney painful, tender, or palpable.

Urine.—Specific gravity 1020, acid. No abnormal elements, chemical or bacteriologically.

Plain X-ray.—Normal-looking kidney shadows. No opacities from calcification.

Cystoscopy.—Ureteric orifices normal on both sides. Some bulging inwards of the posterior bladder wall.

Pyelogram (abrodil—half strength 6 c.c.).—Left pelvis and ureter normal in size and position. The upper major calix is interrupted in its course between pelvis and minor calices by a

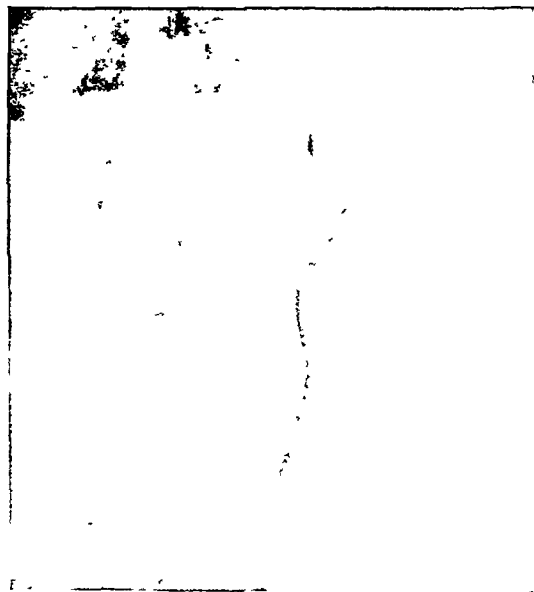


FIG. 7.—Case 1. Pyelographic appearances.

broad expansion in which blotches and patches of shadow appear here and there. At the top of this area are two well-defined and normal minor calices. The middle calix ends abruptly and a faint ribbon of shadow passes upwards from it behind the blotchy area of the upper calix. The lower calix divides into two branches. The upper of these is normal with a minor calix showing at its extremity. The lower branch is somewhat club-shaped and wider than usual. Two normal minor calices appear at its outer termination. The shadow in this lower major calix is uniform, but perhaps a little fainter than that in the main part of the pelvis. (Fig. 7.)

The intravenous pyelograms demonstrated good and equal function of both kidneys, but no pelvic abnormality was apparent.

DIAGNOSIS.—Hydatid cyst of upper pole of the left kidney. The blotchy area was considered to be the cavity of the cyst communicating with the upper calix. No explanation of the normal minor calices opening into the cyst could be suggested until the specimen was examined after nephrectomy.

OPERATION.—Left nephrectomy through a loin incision extended well forwards. No cyst was apparent until the upper pole was reached, when a whitish area was seen at the upper and lateral angle. Dense adhesions to suprarenal capsule and diaphragm. A small portion of the suprarenal capsule was removed with the kidney. The wound was closed except for a wick of rubber dam.

POST-OPERATIVE COURSE.—Primary healing, followed by discharge of a little pus tracking from where the adhesions were separated. Discharged on the twentieth day, with final healing a few days later. In good health after three months.

Comment.—The case up to this point displayed the usual features of an open hydatid cyst of the kidney. The chief interest lay in the examination of the specimen. This showed:—

1. That daughter cysts were present in the middle calix, the pelvis proper, and the ureter, although for three weeks before the operation there had been no colic, pain, or tenderness in the kidney or ureter.

2. That there were three definite mother cysts in the kidney: (a) A primary cyst in the cortex of the posterior part of the upper pole, recently ruptured into the middle calix; (b) A secondary but well-established cyst, with intact mother ectocyst, and containing daughter cysts, in the upper major calix; (c) A younger cyst with intact ectocyst and containing daughter cysts in the lower major calix.

3. That the urine from the upper and lower poles was conducted to the pelvis by normal papillæ which entered the upper and lower major calix discharging between the ectocyst and the pericyst, the latter in this case being the thickened wall of the calix.

4. That the lower cyst was so deeply situated within the kidney that its presence was discovered only after complete section of the organ.

The importance of such findings in a woman who had been relatively free from symptoms for twenty years, and who had no doubt carried the parasite for thirty, warrants a more detailed description of the findings.

DETAILED DESCRIPTION OF THE SPECIMEN.—

External View (Fig. 8).—The kidney was not enlarged and appeared normal, except for the portion overlying the cyst. The latter projected very little from the surface. From the front it was seen as a whitish area with tags of separated adhesions attached, occupying the upper and lateral corner. From behind, a rather larger area was involved reaching almost to the middle of the organ in a downward direction, but affecting only the lateral half. The medial half of the kidney including the upper margin was not involved.

The specimen was bisected in the usual way (Fig. 9). The posterior half showed two cysts, one in the upper pole, and one in the lower, quite unconnected with each other. The mother ectocysts of both were intact. Each contained many daughter cysts of the nearly uniform size of large peas. Free in the pelvis lay two cysts, one at the pelvi-ureteral junction. They were both collapsed. At the lower part of the stump of the ureter just above the ligature and 3 in. from the pelvi-ureteral junction lay a third collapsed cyst adhering loosely to the wall of the ureter. A fourth cyst could be seen peeping from the middle calix, which had not been fully exposed with the section.

Mother ectocyst and daughter cysts were removed and the condition shown in Fig. 10 revealed.

The upper cavity had rugged fibrous walls, which varied from 1 to 2 mm. in thickness and showed yellow patches of calcification. Above, it reached the kidney surface. In front, two normal papillæ serving the kidney cortex of the anterior part of the upper pole projected

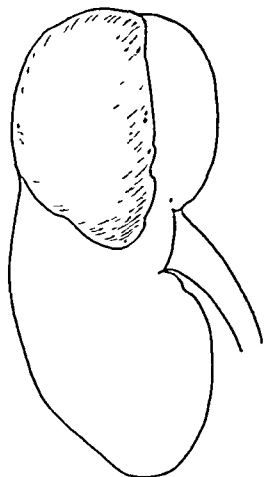


FIG. 8.—Case 1. Diagram showing the extent of involvement of the kidney by the cyst (posterior surface).

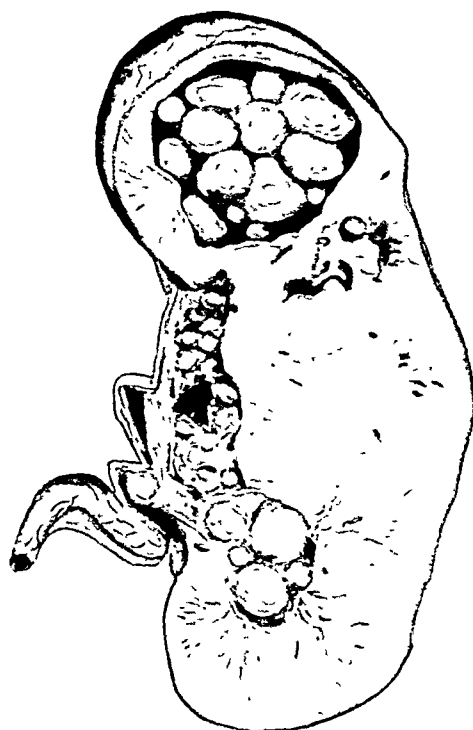


FIG. 9.—*Case 1.* Kidney sectioned showing the two calix cysts with their contained daughter cysts. Both ectocysts are intact. The small cyst seen in the pelvis has come from the middle calix, the upper corner of which may be seen just below the cyst of the upper calix. Papillæ may be seen in relation to both cysts.

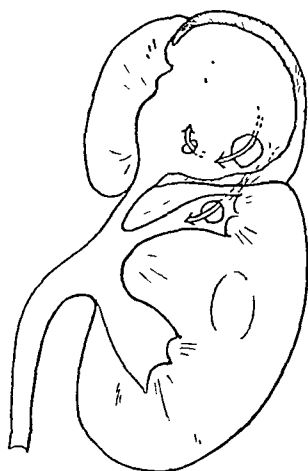


FIG. 10.—*Case 1.* Diagram showing the two orifices of communication between the cortical cyst and that of the upper calix. The lower arrow shows the route by which the contents of the cortical cyst reached the middle calix.

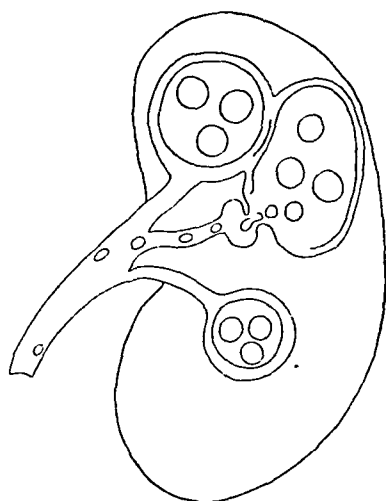


FIG. 11.—*Case 1.* Schematic drawing of the arrangements found in the specimen. The upper and lower calix cysts are closed. Daughter cysts are seen escaping from the middle calix.

into it. They had abutted on the ectocyst, and the urine from them had passed down between it and the adventitia to reach the pelvis proper. The cyst was clearly the upper major calix. Posteriorly, this cavity, which served the dual rôle of calix and cyst, communicated with another cystic cavity through two orifices, well-defined oval rings, occupying the position where normally the papillæ serving the posterior part of the upper pole emerge into the upper major calix. These orifices had all the appearance of being the fibrous rings of the fornices from which the papillæ had been absorbed.

The cavity into which they led was an excavation of the cortex. It was filled with loose daughter cysts of various sizes from clear tense pearls to collapsed pea-sized cysts. A crumpled mass of membrane, probably the shrunken mother ectocyst, lay in a recess (*Fig. 11*). This was the primary cortex cyst. Its contents were prevented from passing into the calix cyst by the tension of the cyst contained in the latter, which occluded the orifices of communication. The cyst wall reached the posterior surface and projected slightly from it. It was thick and fibrous, and in parts calcified, similar to the walls of the calix cyst. Below, it had undermined the base of the papillæ which belonged to the middle calix. One of these papillæ was a mere shell containing a cyst with clear fluid (not hydatid), another had been completely absorbed, and through the fibrous ring of its fornix the cortical cyst communicated freely with the middle calix. The latter was stuffed in every corner with small intact and larger collapsed cysts one of which was projecting into the main pelvis. It was clear that the rupture into the middle calix had provided the material which the patient had passed. These two upper cysts with their thick rugged and partially calcified walls were of undoubtedly long standing.

Lower Calix Cyst.—This was much younger than the other two cysts, though the presence of daughter cysts indicated that it was not a thing of yesterday. The epithelial wall of the calix was little thickened, though white and sclerosed. Normal papillæ from the lower pole projected into the lumen, the conditions being precisely the same as in the upper calix. Two of the cysts, those in the calices, illustrated the condition, which for lack of a better term, I have called 'pseudo-closed'. The pericystic cavity communicated with the pelvis, the endocystic cavity was closed off. The cortical cyst had belonged to the same class, the pericystic cavity communicating both with the upper and possibly with the middle calices. It had, however, recently become an open cyst by rupture of the pericyst or ectocyst into the middle calix.

The puzzling feature of the pyelogram received its explanation. The indefinite blotchy area was not abroad in the cyst, but in the narrow pericystic cavity between ectocyst and calix wall. By this route it reached and defined the two upper minor calices.

The uniform filling of the lower pole cyst indicated a wider and looser space between ectocyst and calix wall obscuring the appearance of filling defect. The ribbon of shadow from the middle calix led to the ruptured cortical cyst.

PATHOGENESIS.—The series of events may be reconstructed as follows:—

Lodgement of the hexacanth in the cortex of the posterior part of the upper pole.

Absorption of secreting tissue as far as the capsule behind, and the base of the upper papillæ in front.

Absorption of the papillæ, leaving the fornix rings intact.

Rupture of ectocyst through the orifices thus produced, causing renal colic with the passage of cysts twenty years ago.

Retention and growth of one cyst or scolex in the upper major calix.

Reconstruction of the original cyst from retained daughter cysts, scolices, or germinal layer elements. It is doubtful whether a new mother ectocyst was formed or not. Many of the small cysts appeared to be loose. Further egress to upper major calix would become shut off by tension of the wall of the new cyst growing there.

Undermining by cortex cyst of papillæ of the middle calix and eventual discharge of contents through the fornix ring and down the ureter, accounting for the recent attack.

The presence of the lower calix cyst is readily explained. In pelvic systole urine is forced back into all the calices as well as down the ureter. A scolex had at one time or another passed into the pericystic cavity of the upper calix cyst and been forced into the lower calix. If its hooklets were extruded, it might be entangled there while its fellows were swept out in the urine.

Several facts of interest emerge from the study of the case.

1. Hydatid cysts may flourish although part of the ectocyst is constantly bathed in urine.

2. They may gain sufficient nutriment by osmosis from a loose adherence to the epithelial wall of a calix, getting the necessary serum elements from the blood and excluding toxic ones from the urine.

3. The presence of a cyst in the calix does not necessarily cause any disturbance of the function of the papillæ or surrounding cortex.

4. The remarkable toleration of the calix to the foreign body.

Probably the vague abdominal pains for which the patient was operated on were due to mild calix colic, but for the last ten years even this symptom had failed.

MICROSCOPICAL EXAMINATION (*Fig. 12*).—Sections were cut through the wall of each cyst. In the upper calix cyst most of the transitional epithelium was missing, though strangely enough part of the adventitia of the cortex cyst had been epithelialized, presumably by continuity through the communicating orifice. The epithelial lining and muscular layer of the

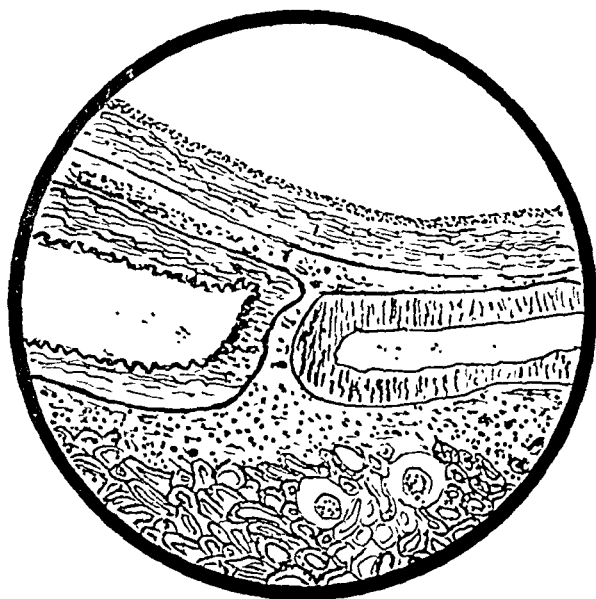


FIG. 12.—*Case 1.* Low-power view of section through the wall of the lower calix cyst. The transitional epithelium lining the calix is intact. Two sections of a cut artery are seen in the middle of the pericyst

lower calix was preserved. The following layers could be demonstrated in all three specimens from within out: (1) Shreds of hyaline laminated membrane with adherent cells, as if from the germinal layer adhering to the inner surface of the cyst wall; (2) Epithelial layer missing in parts; (3) In the lower calix cyst the muscular layer of the lining membrane of the calix separated by loose areolar tissue from the rest of the pericyst; (4) Cellular fibrous tissue containing large blood-vessels; (5) Dense fibrous zone; (6) Advancing zone of fibroblasts with many plasma cells and eosinophils; (7) Kidney tubules and glomeruli.

Processes from the inner fibroblasts could be seen inserting themselves between tubules and the individual cells of the latter. These cells could still be detected singly and in groups in the midst of the advancing fibrous tissue.

Case 2.—Male, aged 32. Clerk.

HISTORY.—The patient lived as a child in the country. Referred for discharging lumbar and abdominal sinuses of twenty-three years' duration. From the age of 5 he had suffered from intermittent attacks of left renal pain. Four years later an abdominal tumour was discovered. Laparotomy was performed and an hydatid cyst of the lower pole of the left kidney cleaned out and marsupialized. Urine flowed from the resulting sinus for some years, but gradually the discharge became pure pus. Several later attempts made to remove the kidney

were unsuccessful and added a loin sinus to the existing abdominal one. Lately the patient has become weak and ill and subject to septic sores on arms and legs.

ON EXAMINATION (Jan. 28, 1924).—Cystoscopic and X-ray examination showed that the left ureter was occluded, the kidney functionless, that the sinus communicated with a pus cavity in the depths, and that the two sinuses together encircled the colon.

OPERATION.—A fibrolipomatous mass containing a cavity corresponding to the original renal pelvis was removed by sharp dissection. This mass was all that was left of the kidney.

SUBSEQUENT HISTORY.—A faecal fistula due to local damage to the colon's blood-supply took nine months to heal. Eleven years after the operation the patient remains well with firm scars and no evidence of hydatid disease.

Case 3.—Female, aged 18. Single.

HISTORY.—The patient was a country girl and had had no previous symptoms. She came to Wellington to be confined. Primipara. Normal labour. On the fifth day of the puerperium she developed severe radiating pain starting in the right loin. Urine became turbid and some membranous material was passed by the urethra. This proved on microscopical examination to be hydatid membrane.

ON EXAMINATION (March, 1924).—On examination twenty-four hours after she had passed the membrane, the urine was clear and contained no hooklets or scolices. The only physical sign was some tenderness on pressure in the right costovertebral angle.

DIAGNOSIS.—Hydatid cyst of the right kidney. The patient was asked to report for detailed examination after she was up and about. However, she left the maternity home, got married, and was not seen again.

Case 4.—Male, aged 52, labourer. Admitted to Wellington Hospital, Nov. 5, 1935.

HISTORY.—Chronic cough for eighteen months, with loss of weight. Has never had urinary symptoms.

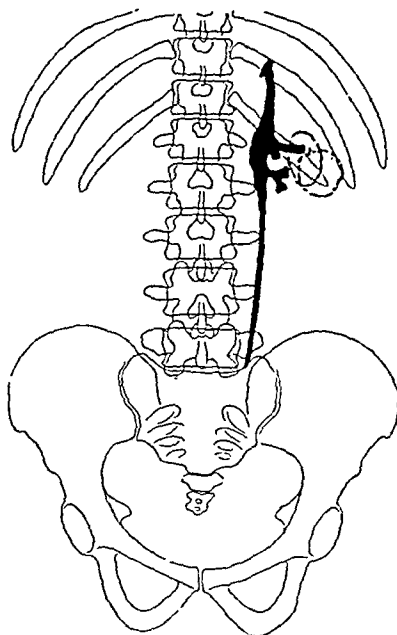


FIG. 13.—Case 4. Diagram of the pyelogram.

ON EXAMINATION.—Diminished breathing over the lower lobe of the right lung. No dullness. Breathing shallow but vesicular. Chest movements poor. No abdominal findings except that on deep inspiration, a tumour the size of a small orange could be felt in the left hypochondrium. It had the characteristic feel of a kidney tumour and moved freely on respiration.

X-ray Examination.—There was no evidence of hydatid cyst in the lungs, the radiograph tending to confirm the provisional diagnosis of carcinoma of the lung. Behind the twelfth rib on the left side at the outer border of the kidney shadow appeared two intersecting ring shadows typical of calcified hydatid pericyst.

Urine.—Trace of albumin, a few erythrocytes, pus cells, and granular casts. No hydatid elements.

Special Hydatid Tests.—Casoni, positive; Ghedini (complement-fixation), strongly positive. Eosinophils, 2 per cent.

Pyelogram of the Left Kidney (Fig. 13).—The lower major calix was normal. The upper and middle major calices formed a right angle with each other, giving the impression that



FIG. 14.—Case 4. Posterior aspect of the left kidney showing the two cysts.



FIG. 15.—Case 4. Anterior aspect of the same kidney. All the cyst contents have been removed. Windows have been cut in both cyst walls but have been replaced in position.

a cyst was prising them apart. They were somewhat dilated and lengthened and the end of the middle major calix almost reached the circumference of the outer of the two ring shadows.

DIAGNOSIS.—As no hydatid material was found in the sputum at any time and there was no other evidence of hydatid disease of the lung, the hydatid cysts were looked on as an incidental finding. It was in doubt whether the cysts were in the spleen or kidney, but while the pyelogram was not pathognomonic a provisional diagnosis was made of a calcified cyst of the outer border and another cyst of the upper part of the left kidney.

COURSE.—The patient died on Nov. 21, 1935, from terminal pneumonic symptoms.

AUTOPSY.—The three important points which were revealed were: (1) Death was due to cancer of the lung; (2) There were two primary cortical hydatid cysts in the left kidney; (3) There was no evidence of hydatid infestation elsewhere in the body.

THE SPECIMEN (Figs. 14-16).—The kidney and the cysts together formed an almost quadrilateral mass 5 in. long by 4 in. wide. One large cyst oval in shape and measuring $8 \times 5 \times 5$ cm. occupied the upper and lateral part of the kidney, reaching rather more than half-way down. It had displaced the upper major calix medially, and the middle major calix inferiorly, so that these two channels formed a right angle with each other as seen in the pyelogram. The lower cyst was more irregular in shape. Below, it formed a circular cavity attached by a wide pedicle to the lower part of the posterior border. The cyst had grown upwards, forming a tapering projection which lay lateral to the large upper cyst. Its medial wall was thus the lateral wall of the latter. Neither the endocystic nor the pericystic cavity of the upper cyst communicated with the kidney pelvis, but there was a small communication between the pericystic cavities of the two cysts about $\frac{1}{2}$ cm. in diameter. The pericystic



FIG. 16.—Case 4. The sectioned kidney—posterior half. The knife has passed precisely through the opening between the two pericystic cavities. The upper toothpick is laid across this opening. The lower shows the direction of the middle major calix thrust apart from the upper major calix so that the two form a right angle.

cavity of the lower cyst communicated freely with the middle major calix, but the contents of the endocystic cavity did not escape, the ectocyst being intact. The ectocyst of the upper cyst was also intact so that the contents of the two endocystic cavities did not mingle. When a pyelogram of the excised kidney was made, slight pressure resulted in the rupture of both ectocysts and the fluid penetrated in such a way that the daughter cysts which filled both cavities were clearly outlined.

Comment.—The presence of two separate primary cortical cysts is rare. The presence of blood-cells in the urine is an interesting and common finding in hydatid cysts of the pseudo-closed variety in spite of the total absence of urinary symptoms. The heavy calcification of the cyst walls, together with the size that the cysts had obtained, indicated that infestation had taken place many years before.

GENERAL OBSERVATIONS ON HYDATID DISEASE OF THE KIDNEY

In discussing the various aspects of the disease, reference will be made to illustrative cases from the literature by the insertion of the name of the author reporting the case in brackets, followed by the number of the case where the article referred to describes more than one. The location of the full report may be found by reference to the author's name in the bibliography.

History.—The first case in which hydatid cysts were passed in the urine was reported at the end of the seventeenth century by a London surgeon, Davies. Earlier in the same century Redi (1626–97), the Milan biologist, had established the parasitic nature of hydatid disease. In 1861 Béraud in his thesis initiated the systematic literature on renal echinococcosis.

Etiology.—The etiology is that of hydatid disease in general. The industry of sheep-raising on the one hand, and insanitary conditions of life on the other, contribute chiefly to its incidence and spread. The former factor prevails in Australia, New Zealand, and the Argentine, the latter notably in Greece and Dalmatia (Toole, Račić).

Pathology.—Renal cysts may be classified pathologically as : (1) *Primary* : derived from a hexacanth embryo. (2) *Secondary* : from a scolex, piece of detached germinal layer, brood capsule, or daughter cyst from some other part of the host. (3) *Invasion* : penetrating the kidney from an adjacent organ. Secondary cysts may be further subdivided into : (a) Embolic—conveyed by the blood-stream ; (b) Calix—sown after rupture of the primary cortical cyst.

1. Unlike the liver, the kidney is almost invariably the site of one primary cyst only. Račić records a case in which a kidney containing eight cysts, each with daughter cysts, was removed, with a clinical cure. These were, however, almost certainly secondary embolic cysts. Frequently there are calix cysts in addition to the primary cortical one. In many cases at post-mortem examination the renal cyst is the only one in the body. However, others, dead or alive, may be found in the liver or other organs. Toole found the kidney cyst the only one in 42 out of 46 cases, Nicaise in 416 out of 449 cases. Some of these, however, were purely clinical findings. Other cysts may well have been missed, more especially in the pre-radiographic era.

2a. Secondary embolic cysts are usually multiple and accompanied by cysts elsewhere. Even in the latter case one kidney may be spared (Peacock and Wale Hicks), but both are more commonly involved (Welsh and Chapman, Kinyon, Donovan). While secondary cysts from a ruptured liver or spleen may attach themselves to the peritoneal surface of the kidney (Allaben), there is no authentic record of invasion of the organ. Rupture of a cyst into the heart cavity is the usual cause of metastatic spread. Bocker found in one such case that the left renal artery was blocked by a daughter cyst. Liver cysts may, however, be associated with multiple secondary cysts of the kidney, the lungs remaining free.

Secondary renal cysts are rare compared with primary (25 out of 474 cases in Nicaise's series). They may contain daughter cysts, rupture, and behave exactly like the primary ones.

2b. Calix cysts are well illustrated in *Case 1*. They are always secondary to cortical cysts. Primary cysts of the renal pelvis are unknown. Of the two

mentioned by Nicaise, one (Bland-Sutton) was an error in translation, the other an unverified personal communication. Calix cysts as a rule are small and discoverable only by section of the excised kidney. Only one has been hitherto recognized as such (Willans and Stewart). Other illustrations leave little doubt that the secondary cysts were in one of the calices (Lutz, Livermore, Vila, and probably Filienz and Melen). In cases of complete kidney destruction the pelvis may be detected as a separate slit-like cavity or it may contain the cyst. The latter condition is probably the end-result of a calix cyst.

3. Invasion cysts in which liver or spleen as well as kidney are involved (Craig and Lee-Brown, *Cases* 9 and 10, E. L. Young, *Case* 2) leave some doubt as to the organ primarily affected. The pyelographic evidence is in favour of the kidney as a rule. Alivisatos, however, reports an undoubted case of rupture of a liver cyst into the right renal pelvis.

Echinococcus Alveolaris.—Only two kidney cases of this curious variant have been reported, those of Sabolotnow in 1867 and of Hahine in 1928. In Hahine's case the nodule in the kidney was secondary to one in the liver. It is still maintained by Posselt, of Innsbruck, that this variety constitutes a separate species. Evidence is accumulating against it. Dévé recently abandoned the dualist theory in an article entitled "Transitional Forms between Hydatid Echinococcus and Alveolaris" in which Dew's Australian case of *E. alveolaris* was cited. Posselt, however, returned to the attack with the uncompromising title, "There are no Transitional Forms". There the matter rests, and in any case the alveolar form in the kidney is a mere curiosity and requires no further mention.

Side Affected: Age and Sex Incidence.—In the larger series of cases both kidneys are equally affected and bilateral cysts are rare. Nicaise found 197 with the right kidney involved, 185 with the left, and 12 bilateral. The sexes are equally vulnerable.

Most cases are infected in childhood; the third to the fifth decades are the commonest periods for the development of symptoms. In the reports the youngest patient operated on was a child of 2 years (Schepel).

The percentage of kidney cases to all hydatid ones ranges from below 1 per cent in Argentine statistics to 4.65 per cent in Račić's series in Dalmatia. Compared with other surgical diseases of the kidney echinococcosis is very rare. Many urologists with large practices have never seen a case. The largest personal experience was that of Craig and Lee-Brown.

Site of Implantation.—The commonest site is the cortex of the anterior surface of the upper and lower poles. Cysts of the posterior surface as in *Case* 1 are rare. The cyst may arise in the loose tissue of the sinus and be adherent to the pelvis of the kidney (Bland-Sutton, Guiteras, Spurr, Rowlands, Keyes and Busch, Vedenski, 4); in the middle portion, bisecting the kidney and leaving the two poles intact (Fillenz, Račić, 10, Vedenski, 3); or it may occupy the whole kidney, only a minute nodule of renal substance remaining (Staveley, Marsh, Russell and Kilbane, Meltzer, Craig and Lee-Brown, 10, Leighton and Lewis).

The cyst may be very superficial, almost pedunculated (Erdélyi, Woodman, 2, Stark, Flynn, Geyman, Bressot). It may be so small as to defy detection until the kidney is completely split open (Craig and Lee-Brown, 16) or so large as to fill most of the abdomen (E. L. Young, 2, Craig and Lee-Brown, 2, Vedenski, 5, and Marsh). In Marsh's case the cyst measured $29\frac{1}{2}$ by 13 in. and weighed 10 lb.

Development and Ultimate Fate of Parasite.—In the kidney, as elsewhere, the hydatid cyst develops very slowly, though growth may be stimulated by trauma or pregnancy. Expansion is centrifugal but the centre point of the sphere is not static. As a rule the line of least resistance is followed, but in the effort to preserve its circular contour the cyst may burrow into the spleen (Craig and Lee-Brown, 10), into the liver (Craig and Lee-Brown, 9, E. L. Young, 1), or the tail of the pancreas (Perrin). A fibro-cellular proliferation—the pericyst—acts as advance guard, the fibroblasts sending their processes between individual tubules, then between the cells, which appear as islands in a flood before being finally overwhelmed and absorbed. The outer circumference of the cyst, once it has reached and passed the kidney capsule, makes rapid progress into the perirenal tissues or bulges into the peritoneal cavity. Simultaneously the inner circumference may reach the calix wall, which offers resistance. The papillæ, being of soft tissue, are first absorbed. The pericyst may remain intact, or, left in the air, lose blood-supply and necrose. In the latter case the cyst becomes one of the pseudo-closed group and the ectocyst, though still intact, presents directly into the lumen of the calix. It may, however, hold for a long time, as the inner tube of a motor tyre may hold in spite of a small defect in the outer cover. The communication of the pericystic cavity with the calix may set up irritation and produce calix colic. Finally, owing to extra pressure, trauma, or strain, the ectocyst itself bursts and the contents pass through the fornix ring into the calix and down the ureter. This appears to be the sequence of events of the rupture of the cyst into the pelvis. Sometimes an escaped daughter cyst or scolex takes root in the calix wall and eventually by its growth and tension blocks the hole through which it came as a sail drawn under a ship's hull stops a leak. The scolex may be conveyed, by the current of urine, to a distant calix and grow there. The primary cortex cyst continues to grow and may eventually burst into yet another calix (*see Fig. 11*). Trauma may cause rupture of the ectocyst (Ivanov, Grove), or pregnancy or labour pains may determine the final break (*Case 2*, above, Vedenski, 3, Cowan, Flynn, Craig and Lee-Brown, 5). Even simple abdominal palpation may produce the same result (Mensikov).

The escaped cysts and hydatid debris may lie in the pelvis and ureter and cause no discomfort, or there may be violent renal colic, eventual dilatation of the ureter, and even hydronephrosis from obstruction of the duct. The cysts may stay in the bladder, causing strangury, difficulty, and retention.

The hydatid may die of old age or lack of nutriment. There is a ring of large blood-vessels in the pericyst, a striking microscopical feature (*see Fig. 12*). Thus ample serum is brought to the hyaline laminated membrane from which the parasite can select its food supplies. Calcification hinders the osmotic process. The animal feels the urge for more defensive propagation. Daughter cysts are born in large numbers and even granddaughter cysts (Farkas). Tension may become low and growth in abeyance or spasmodic. The whole tribe may perish and be buried like criminals in lime, or sepsis may supervene with unpleasant results both for guest and unwilling host. Low-grade life may, however, be maintained even though the pericyst is heavily calcified. Daughter cysts resist sepsis for a considerable time (Bird). The cysts may be sterile, or, though univesicular, contain brood capsules and scolices (Schmidt, Vedenski, 4 and 5). Far more frequently they are full of daughter cysts at the age period when the patient usually becomes sensible of unpleasant symptoms. Propagation is by endogenous budding, but the Russian

workers Massalitinov, Napalkov, and Keropian consider that reproductive elements in the pericyst are the chief cause of recurrence after operation. It is true that in confined spaces, as in bone, exogenous budding takes place by herniation (Dew), but it seems more probable that the fertile causes of recurrence are the pieces of the ectocyst that adhere to the pericyst even when the former appears to be loose and easily removable (Kaïris).

Complications.—The expanding pericyst may become incorporated with the stomach, spleen, pancreas, colon, liver, duodenum, or vena cava. The adrenals may become inseparably fused. Such a welding of tissues becomes a danger at operation, chiefly where the surgeon confuses it with ordinary adhesion and attempts to separate the inseparable. There is no reason why a large upper pole cyst should not rupture into the bronchial system. The five reported cases are, however, unconvincing (Nicaise 2 cases, Heer, Nicolich, and Christie and Fawcitt). In one of these at least a cyst was found subsequently in the lung. Empyema by lymphatic spread of sepsis may occur, or from bacteria accumulating outside the laminated membrane like debris on a filter paper. Abdominal cysts may rupture into the peritoneum. Such a case, as yet unpublished, exists in the Registry of Hydatids kept by Sir Louis Barnett for the Royal College of Surgeons of Australasia. Another ancient case is reported by Nicaise. I can find but one solitary report of rupture into the stomach, a fatal complication (Nicaise), and three into the colon, with amelioration of symptoms. One of these three, however (Parlavecchio), proved to be a pararenal cyst associated with another abdominal one. It was the latter that discharged into the colon. Sudden death from rupture into the vena cava such as has occurred with liver cysts (Pitts, K. D. Fairley) has not been reported. Indeed, the opening of a kidney cyst into any other viscus than the kidney pelvis is so rare that the possibility may almost be discounted in estimating the prognosis. Embolic spread from the kidney is also so rare as to be negligible.

Complications within the urinary tract include hydronephrosis, pyonephrosis, lowering of kidney capacity on both sides from difficulty in micturition or chronic retention due to accumulation of membrane in the bladder, with or without bilateral ascending pyelonephritis. All are rare and mostly due to sepsis. The ureter on the affected side may become completely occluded (*Case 2* above, Haynes, Lutz). In the four recorded cases of solitary kidney with hydatid infestation, kidney function was well maintained (Blackburne, Houzel, Nicaise, 2). Multiple cysts may kill from uræmia through destruction of kidney substance (Kinyon, Donovan) or by an associated abdominal cyst or cysts blocking the ureter of the unaffected kidney. A perinephric abscess may develop and point in the loin.

Associated Pathology.—Calculus associated with hydatid cyst is rare even in such countries as Dalmatia where both conditions are common. Nicaise in his large series reports only twelve cases and includes stones in the bladder. Other cases are those of Vedenski, 4, Craig and Lee-Brown, 16, Blackburne (in a solitary kidney), Castaño and Astraldi, and an unpublished case in the R.A.C.S. Registry. I can find no other examples. Other instances of associated pathology are: malignant growth (Erdélyi), floating kidney (Eberle, Celowski), ectopic kidney (Juvara), adenoma (Galvez and Garate), and carcinoma of the bladder (Thomas). Ferruccio had a case of associated Addison's disease, but it is not clear whether this was due to adrenal involvement. Cancer may develop in a marsupialized cyst (Bonamy).

Symptomatology.—No symptoms at all may appear for many years, and the patient may be quite ignorant of the existence of the cyst till the day of his death. Closed cysts may be found accidentally in perfectly well individuals. The parasite may produce unexplained anaphylactic phenomena such as urticaria or obscure pressure symptoms. Those that grow towards the abdomen cause indigestion, pain, and fullness after meals by encroaching on the stomach, or diarrhoea, constipation, or even complete obstruction by pressure on the colon. In rare cases a gradual loss of strength, weight, and energy with ultimate symptoms of uræmia may result in death and the cause be discovered only at autopsy.

Pseudo-closed cysts cause the intermittent localized pains of calix colic and urinary symptoms of frequency and hæmaturia, accompanied by painful micturition and urgency if sepsis has supervened. All symptoms may be initiated or augmented by trauma, pregnancy, or labour.

The rupture of the ectocyst may be heralded by a sudden excruciating attack of pain in the loin or upper abdomen with extreme tenderness and rigidity of the abdominal muscles. Accentuation of the symptoms may be due to anaphylaxis added to spasm of the renal pelvis and ureter. Such cases have more than once been subjected to immediate laparotomy with a view to the repair of a supposed perforated gastric or duodenal ulcer. The very first symptom may be retention of urine due to an accumulation of membrane in the bladder. The temperature may be raised and the pulse rapid even when there is no sepsis, especially during or following attacks of renal colic. In more than half the cases a mild attack of colic is followed at the end of twenty-four or forty-eight hours by some difficulty in micturition and the eventual passage of membrane or daughter cysts.

The attack may never recur or may be repeated at intervals of a month, a year, or even many years. The onset of severe sepsis modifies the subsequent symptoms. In its absence the renal colic may become less severe with each attack owing to dilatation of the ureter, whereas the difficulty in emptying the bladder may be greater owing to the increasing abundance of hydatid material which reaches it. Between attacks the general health is, as a rule, excellent, and there is complete freedom from symptoms. The onset of infection may convert the intermittent loin pain into a constant nagging ache and there may be superadded strangury, frequency, scalding, and later the evidence of bilateral pyelonephritis in loss of appetite and general debility.

Diagnosis.—In open cases diagnosis is made from the examination of the material passed. If this is only 'hydatid sand' or brood capsules, the case may be considered merely a 'gravel' one, and the patient has perhaps to wait some time before a microscopical examination of the sediment discloses hooklets and scolices (Huffman). Calcified cysts may produce solid debris which when passed is mistaken for calculus (Croveri and Placeo). Retrovesical hydatid cysts fully reported on by Deeming very rarely rupture spontaneously into the bladder. I have found five records only (Nicaise, Fairbanks, Compan, Craig and Lee-Brown, Ortiz and Garcia). An additional case in which one of a group of multiple secondary cysts opened into the bladder is on record in the R.A.C.S. Registry. Confusion of such cases with renal echinococcosis is most likely when the symptoms of the latter are confined to bladder difficulties with no renal pain or colic (Vedenski, 1 and 2). Cystoscopy with pyelography should clear up the diagnosis. It was the fortunate lot of two surgeons, Blum and Pereschewkin, actually to see a daughter

cyst extrude itself from the ureter. Craig was able to pass a cystoscope into a retro-vesical cyst. The bare possibility of a liver cyst rupturing into a kidney pelvis (Alivisatos) must be remembered.

Pseudo-closed cysts are demonstrated by typical pyelograms. Closed cysts are the most difficult. Clinically they appear as tumours in the hypochondrium or free and mobile in the abdominal cavity (Račić, 10). If the muscles are rigid, such a tumour may be missed during an attack of renal colic. An hydatid tumour feels solid. If fluctuation is detected some other pathology is more likely (Bird). Hydatid 'thrill' ('Schwirren', 'frémissement'), well described by Barnet, is so rare as to be valueless in most cases. The conditions required are a few large daughter cysts, fluid under low tension, a thin abdominal wall, and contact of a daughter cyst with that wall of the mother cyst which lies under the examining hand. The sensation is of a delicate metallic spring vibrating, accompanied by a sonorous resonance on auscultation. The 'thrill' in renal cases was detected in the cases of Burburg, Peyrot, Spurr, and Christie and Fawcitt.

The commonest errors in tumours appearing in the hypochondrium are to mistake the cyst on the left side for an enlarged spleen, especially where malaria is endemic, and on the right side for a liver cyst. In the former the special serum tests, with X-ray examination and pyelograms, usually clear up the diagnosis; in the latter serum tests are useless.

An upper pole cyst may produce high dullness at the lung base of the corresponding side and has to be distinguished from an intrathoracic condition or a splenic cyst as well as from a hydronephrosis. The greatest calamity in such cases is a diagnostic puncture.

Where there are no physical signs at all, routine urological examination may show deformity in the kidney pelvis and the serum tests eliminate other confusing renal conditions. In cases of neoplasm of the kidney, the ureter is often kinked, in cysts it maintains its contour (von der Becke). Any abnormality may, however, exist in the kidney and the serum tests be positive on account of hydatid in the liver.

X rays may show a density due to the high saline content of hydatid fluid or a typical curvilinear shadow of a calcified pericyst. In all types of case complete urological investigation usually gives fairly conclusive information, but the details of the cystoscopic and pyelographic findings are such that they can be treated effectively only at length, and in a separate paper. Abundant material for this purpose is now available from the cases of the last ten years.

Serum Tests.—The precipitin test, first put on an effective basis by the work of Fleig and Lisbonne, Welsh and Chapman, and others, is rarely used. The complement-fixation test, worked out by several separate workers, notably Ghedini, Imaz Apphatie, and Weinberg, between 1906 and 1908, is a laboratory proceeding requiring rigid conditions of technique to give its best results. The intradermal reaction of Casoni, taking into account both the immediate and delayed reactions, is simple, practical, and of great value. For the technique, interpretation, and use of these tests the reader is referred to the published writings of the workers in that field. Among the most prominent in the southern hemisphere are Imaz Apphatie, Nuñez, Dew, K. D. Fairley, N. H. Fairley, Kellaway, Williams, and Hercus.

Prognosis.—From the statistics available hydatid disease of the kidney appears on the whole to be relatively innocuous. Of the 474 cases of Nicaise 20 per cent were routine autopsy findings: 215 cases received no surgical treatment, and only

16 died as the direct or indirect result of the cyst. The first attack of colic and the passage of membrane may be the last in spite of an active parasite. Impairment of renal function to a dangerous level is rare even in old-standing cases, and the general health is seldom affected. Spontaneous cure results in many cases (Goinard), though symptomatic cure is probably more common than anatomical.

Prevention.—Preventive measures for hydatid disease in general are retarded by the increased overhead cost added to the industry of wool and frozen meat production, and by ignorance, conservatism, and poverty in the older countries. They consist of the establishment of bureaux and registries, public educational campaigns, and the elimination of the tapeworm in dogs by cooking of all offal fed to them, strict regulations to keep them out of the neighbourhood of abattoirs, and the regular administration of vermifuges. Progress has been made, but results must necessarily be slow in appearing, and indeed will not be fully apparent for a generation, as infection is in childhood and manifestation of symptoms in middle life.

Treatment.—Treatment is mainly surgical or expectant. Turpentine has been used to assist elimination of the daughter cysts (Mackey). No intravenous therapy is effective. Nephrectomy was introduced by Bockel to replace the universal method of marsupialization in 1887. Intervention must be governed by two considerations. In the first place the disease is a benign one, and a cyst once communicating with the pelvis, as a rule, ceases to grow because the level of tension required in the fluid for this purpose re-opens the safety-valve. This rule is subject to modification where a tense calix cyst prevents the re-using of the original portal of communication, as in *Case 1* (see *Fig. 11*). In the second place radical operation is difficult owing to dense adhesions and is not to be lightly undertaken. As a rule no operation should be performed :—

1. After a first attack in which cysts are passed in the urine and investigation shows that there is only a small cyst and the kidney function is good on both sides. The danger of complications is small and subsequent intervention may be undertaken should disabling symptoms or septic complications occur.

2. If the other kidney is absent, infantile, or functioning badly from any cause, and no serious disability is caused by the hydatid. In the best hands any operation on a difficult kidney may lead to damage demanding nephrectomy and the risk is greater than leaving the parasite. Houzel unknowingly removed a solitary kidney.

3. If the cyst is calcified and dead, unless passage of calcified fragments threatens damage to the urinary tract or is seriously disabling.

In most other cases nephrectomy, if possible, is the operation of choice if the other kidney is normal in function, for the following reasons :—

1. The mortality is low, the convalescence short, and health quickly restored.

2. Secondary calix cysts may exist, making recurrence inevitable if the kidney is left. They cannot be detected during any conservative operation.

3. An apparently closed kidney may really be of the pseudo-closed type, and with conservative operations there is not only the danger of recurrence but also of infecting the urinary tract.

4. In many cases the whole of the pericyst cannot be removed, and in these recurrence is common. If the kidney is left, this will manifest itself by the old symptoms of renal colic and the passage of membrane. When the kidney is removed the recurring cyst will cause no trouble, and in the course of many years will reach the surface in the loin where it can be easily dealt with.

When the cyst hardly invades the renal substance at all and has never caused urinary symptoms of any kind, partial nephrectomy with removal of the cyst may be undertaken, but if some of the pericyst must be left, recurrence is likely under any technique and it is better to remove the kidney.

All surgeons agree on nephrectomy if kidney function is markedly lowered, nearly the whole of the organ is occupied by the cyst, or gross sepsis is present. The extreme conservative school go so far as to use partial resection even in open cases (Bengolea), or resect and repair part of the pelvic wall if the cyst is attached to it (Spurr). The only justification, it appears, for such procedures—and it has some validity—is that recurrence is not apparent, because the symptoms of it, owing to the slow growth of the cyst, may not appear in the patient's or the surgeon's lifetime.

In many cases the pericyst is so welded to the surrounding structures that it is quite unjustifiable to endeavour to remove it all. Attempts to do so have led to immediate death from hæmorrhage (Fulton), to tearing of a mesenteric artery with a fatal result (Mensikov), and to wounding the pedicle of the spleen with compulsory splenectomy (Perrin). In such cases the cyst may be cleaned out, formalinized, and left with primary suture of the parietes; or Račić's aseptic marsupialization method may be used. This consists in filling the cavity with antiseptic gauze and not re-dressing till the tenth day. Tube drainage should only be used for pus cases or those in which a pseudo-closed cyst is suspected and it is impossible to remove the kidney. In the latter there will be a urinary discharge following the operation. Marsupialization prevents recurrence by the prolonged sepsis killing both the parasite and the kidney, as in *Case 2* reported above. Vila had to perform nephrectomy on three of his cases for recurrence after conservative operations. It is, however, better to risk recurrence than subject the patient to tube drainage or ordinary marsupialization in non-septic cases.

The ureter should be severed well down to prevent recurrent cysts discharging through it, but complete ureterectomy, even though loose cysts may be left as in *Case 1* and in Willans and Stewarts's case, is unwise. Fresh planes for recurrence are opened up; an operation, in any case difficult, is prolonged; and there is no recorded instance where a cyst has recurred in the ureteral stump.

Operation should be by the lumbar route, with the necessary forward extension and rib resection in difficult cases. The transperitoneal route handicaps the surgeon because there is no direct access for the sharp dissection of adhesions, and apart from other cogent considerations the mortality is higher (32 per cent against 8·5 per cent in the statistics of E. L. Young). In upper pole cases it is sometimes necessary to adopt the transcostodiaphragmatic route.

SUMMARY

1. A general description is given of the echinococcus and the route by which the embryo reaches the kidney, followed by an analysis of the published case reports of the last ten years.

2. Four personal cases are described.

3. The classification of the cysts of the kidney into closed, pseudo-closed, and open corresponds to certain cardinal symptoms.

4. Hydatid cysts of the kidney may be secondary, primary, or invasion.

5. The great majority are primary, but secondary calix cysts may be present and cannot be detected till the kidney has been excised and sectioned.

6. The prognosis is good, as renal cysts rarely rupture into neighbouring cavities or organs, with the exception of the kidney pelvis.

7. Diagnosis should be made in all open cysts and in a large per cent of closed ones by the use of urological methods, X rays, and the special serum tests.

8. The treatment where possible should, as a rule, be nephrectomy, but under certain conditions no operation should be done.

I have to thank Sir Louis Barnett for giving me access to the valuable cases in the Registry of Hydatids of the Royal Australasian College of Surgeons and for assistance in elucidating many points in the life history and habits of the parasite ; also Dr. P. A. Treahy for his kindness in preparing the two coloured illustrations.

BIBLIOGRAPHY

The numbers in brackets indicate the number of case reports in the article referred to if more than one.

* Indicates that the case was a pararenal one.

- ALIVISATOS, N., *Bull. et Mém. Soc. nat. de Chir.*, 1934, lx, 1112.
 ALLABEN, J. E., *N. Amer. Pract.*, 1891, iii, 612, quoted by Kretschmer.
 ANDRÉS, F. PÉREZ (2), *Clin. y. Lab.*, 1932, xx, 226.
 ASCOLI, R., *Osp. maggiore*, 1932, xx, 407.
 BABINI, R., *Gaz. deg. Osped.*, 1929, l, 581.
 BARNETT, L. E., *Surg. Gynecol. and Obst.*, 1927, xlv, 148 ; *N.Z. Med. Jour*, 1921, xx, 277 ; 1934, xxxiii, 191 ; 1935, xxxiv, 1 ; *Jour. Coll. of Surg. of Australasia*, 1929, i, 351 ; *Austral. and N.Z. Jour. Surg.*, 1935, iv, 211.
 BARNEY, J. D. (2), *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
 * BEDARIDA, N. V., *Minerva med.*, 1927, vii, 1330.
 BENGOLEA, A. J., *Bol. y. Trab. d. l. Soc. d. Cir.*, 1926, x, 445.
 BERAUD, E., *Thèse de Paris*, 1861.
 BIRD, F. D., *Med. Jour. of Australia*, 1925, i, 258.
 BLACKBURNE. Quoted by Nicaise.
 BLAND-SUTTON, J., *Tumours Innocent and Malignant*, 5th ed., 652.
 BLUM, V. (4), *Zeits. f. urol. Chir.*, 1933, xxxvii, 46.
 BOCKEL, *Étude sur les Kystes h. du Rein*, 1887. Paris : Alcan.
 BOCKER, A., *Inaug. Dissert.*, 1868. Berlin.
 BONAMY, R., *Paris chir.*, 1910, ii, 100, quoted by Russell and Kilbane.
 BOROSS, E., *Zeits. f. Urol.*, 1926, xx, 534.
 BORRELL, J. H., and BARNES, J. M., *N.Y. State Jour. Med.*, 1933, xxxiii, 1390.
 BRAILSFORD, J. F., *Brit. Med. Jour.*, 1931, i, 133.
 BRESSOT, E., *Lyon chir.*, 1929, xxvi, 73.
 BURBURG. Quoted by Nicaise.
 BURFORD, C. E., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
 CASONI, T., *Fol. clin. chim. et micro.*, 1911-12, iv, 5.
 CASTAÑO, E., and ASTRALDI, A., *Rev. d. Especialid.*, 1926, i, 696.
 CELOWSKI. Quoted by Russell and Kilbane.
 CERVEŇANSKÝ, J., *Čas. lék. čes.*, 1932, lxi, 389.
 CHRISTIE, G. W., and FAWCITT, R., *Brit. Med. Jour.*, 1934, i, 237.
 COMPAN, V., *Arch. franç.-belg. de Chir.*, 1928, xxxi, 509.
 COUNOT, J., *Arch. d. Mal. d. Reins*, 1930, v, 192.
 COWAN, A. F., *Jour. Obst. and Gynecol. Brit. Emp.*, 1929, xxxvi, 592.
 CRAIG, G., and LEE-BROWN, R. K. (16), *Surg. Gynecol. and Obst.*, 1928, xlv, 668.
 CROVERI, P., and PLACEO, F., *Boll. e Mem. Soc. piemontese di Chir.* 1932, ii, 149.
 DAINELLI, M., *Ann. ital. di Chir.*, 1927, vi, 908.
 DAVIES, Quoted by Nicaise.
 DEEMING, C. L., *Jour. of Urol.*, 1923, x, 1.
 DÉVÉ, F., *Thèse de Paris*, 1901 ; *Comptes rend. Soc. de Biol.*, 1913, lxxiv, 735 ; *Ann. d'Anat. pathol.*, 1933, x, 1155 ; *Austral. and N.Z. Jour. of Surg.*, 1934, iv, 99 ; *Comptes rend. Soc. de Biol.*, 1934, cxv, 954.

- DEW, H. R., *Hydatid Disease*, 1928. Sydney: Aus. Pub. Co. *Med. Jour. of Australia* Supp., 1924, i, 113, and 1925, i, 471; *Jour. Coll. of Surg. of Australasia*, 1929, i, 337; *Brit. Jour. Surg.*, 1930, xviii, 275; *Brit. Med. Jour.*, 1935, ii, 620.
- DÓNOVAN, R. E., *Prensa méd. argent.*, 1931, xvii, 1460.
- EBERLE, A., *Zentralb. f. Chir.*, 1911, xxxviii, 1656.
- ERDÉLYI, J., *Fort. a. d. Geb. d. Röntgenstrahl.*, 1928, xxxviii, 858.
- FAIRBANKS, Quoted by Deeming.
- FAIRLEY, K. D., and associates, *Med. Jour. of Australia*, 1922, i, 346; 1923, ii, 27; 1929, ii, 320; *Jour. Coll. Surg. of Australasia*, 1930, ii, 317; *Austral. and N.Z. Jour. Surg.*, 1933, ii, 236.
- FAIRLEY, N. H., *Med. Jour. of Austral.*, 1922, i, 341; *Quart. Jour. Med.* 1922, xv, 244.
- FALIN, D. V., *Vestnik khir.*, 1934, xxxvi, 138.
- FARKAS, I., *Zeits. f. Urol.*, 1926, xx, 534.
- FERRUCCIO, B., *Gaz. deg. Osped.*, 1933, liv, 1307.
- FILLENZ, K., *Zeits. f. Urol.*, 1926, xx, 65 and 96.
- FINSEN, J., *Arch. gén. de Méd.*, 1869, xiii, 23 and 191; *Ugeskr. f. Læger*, 1867, iii, ser. 3, Nos. 5 and 8.
- FLEIG and LISBONNE, *Comptes rend. Soc. de Biol.*, 1907, lxii, 1198.
- FLYNN, R., *Austral. and N.Z. Jour. Surg.*, 1932, ii, 99.
- FULTON, R. A. H., and BARNETT, L. E., *N.Z. Med. Jour.*, 1926, xxv, 317.
- GALL, D. (66), *Ukraine nov. chir. arch.*, 1928, xvi, 564; *Zeits. f. urol. Chir.*, 1930, xxx, 84.
- GALVEZ, I., and GARATE, F. O., *Rev. Assoc. méd. argent.*, 1932, xlv, 622, 803, and 990.
- GEYMAN, M. J., and CLARK, D. M., *Amer. Jour. Roentgenol.*, 1934, xxxi, 541.
- GHEBINI, G., *Gaz. deg. Osped.*, 1906, xxvii, 1616; 1907, xxviii, 53.
- GOINARD, P. (36), *Bull. méd. Paris*, 1930, xlv, 275.
- GREENWAY, D. J., *Semana méd.*, 1922, ii, 761.
- GROVE, H., *Bol. Soc. d. Cir. d. Chile*, 1927, v, 144.
- GUITERAS, R., *Urology*, 1912, i, 496. New York: Appleton & Co.
- GUTIERREZ, A., *Bol. y Trab. Soc. d. Cir.*, 1926, x, 442.
- HAHINE, Z. D., *Russkaya klinika*, Moscow, 1928, ix, 315.
- HAYNES, S., *Ann. of Surg.*, 1902, xxxvi, 95.
- HEER, T. Quoted by Nicaise.
- HERCUS, C. E., *N.Z. Med. Jour.*, 1926, xxv, 325.
- HOUZEL, *Rev. d. Chir.*, Paris, 1898, 689 and 811.
- HUFFMANN, L. F., *Jour. of Urol.*, 1926, xvi, 197.
- HURLEY, V., *Austral. and N.Z. Jour. Surg.*, 1933, ii, 326.
- IMAZ APPHATIE, I. L., *Rev. méd. lat-amer.*, 1932, xvii, 1645.
- IOANIDI, S. S., *Vrath Gaz.*, 1928, xxxii, 1113; *Zeits. f. urol. Chir.*, 1932, xxvii, 216.
- IVANOV, N., *Med. univ.-klin. Voronezh.*; *Zeits. f. urol. Chir.*, 1928, xxvii, 216.
- JEANNENEY, G., *Bordeaux chir.*, 1932, iii, 36.
- JUVARA, E., *Bull. et Mém. Soc. de Chir.*, 1913, xxxix, 103.
- KAIRIS, Z., *Zeits. f. Urol.*, 1927, xxi, 677; *Zeits. f. urol. Chir.*, 1934, xxxix, 22.
- KELLAWAY, C. H., and FAIRLEY, K. D., *Med. Jour. of Australia*, 1932, i, 340.
- KEROPIAN, Monograph, 1930, Krasnodar, quoted by Racić.
- KERSENTE, F., *Prat. méd. du Nord d. l'Afrique*, 1932, v, 622.
- KEYES, W. C., and BUSCH, F. C., *Buffalo Med. Jour.*, 1891, xxxvi, 25, quoted by Kretschmer.
- KINYON, J. J. Quoted by Kretschmer.
- KIRWIN, T. J., *Jour. of Urol.*, 1926, xv, 273.
- KRESTOVSKY, V., *Vestnik khir.*, 1927, x, 169.
- KRETSCHMER, H. L., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
- KRETSCHMER, H. L., (18) *Surg. Gynecol. and Obst.*, 1923, xxxvi, 196.
- KRETSCHMER, H. L., and MOODY, A. M., *Surg. Gynecol. and Obst.*, 1914, xix, 766.
- KUSTER, E., *Die Chirurgie der Harnleiter*, 1896. Berlin. "Tierschmarotzer der Niere", *Deut. Chir. (Bergman and v. Bruns)*, No. 52, 663.
- LABOVITI, V., *Med. Welt*, 1932, vi, 743.
- LANOS, J., *Bull. et Mém. Soc. de Chir.*, 1929, xxi, 628.
- LEE-BROWN, R. K. (2), *Jour. of Urol.*, 1930, xxiii, 611.
- LEIGHTON, W. E., and LEWIS, B., *Amer. Jour. Urol.*, 1912, viii, 71.
- LIVERMORE, G. R., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
- LUTZ, *Zeits. f. urol. Chir.*, 1929, xxvii, 165.
- MACKEY, E., *Lancet*, 1905, ii, 601.
- MACKEVININ, N. E., *Vrath Gaz.*, 1927, xxxi, 260.
- MANUJLOV, A. I., *Omsky Med. Jour.*, 1928, iii, 25.
- * MARGARUCCI, O., *Arch. ital. di Chir.*, 1927, xviii, 447.
- MARSH, J. O., *Cincinnati Lanc. and Observ.*, 1869, xli, 539, quoted by Kretschmer.

- * MASSIMINO, A., and FINOCHIETTO, R., *Prensa méd. argent.*, 1930, xvi, 1576.
 MASSALITINOV. Quoted by Račić.
- MELEN, D. R., *Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 1725.
- MELTZER, M., *Ibid.* 1929, xcii, 1925; *Amer. Jour. Surg.*, 1929, vii, 418.
- MENSIKOV, F., *Sibir. arch. teor. i klin. med.*, 1928, iii, 297; *Zeits. f. urol. Chir.*, 1929, xxvii, 404.
- MOLLO, L., *Policlinico*, 1930, xxxvii, 557.
- MORSON, A. C., *Proc. Roy. Soc. Med.*, 1926-7, xx, pts. 1 and 2, 719.
- NAPALKOV, N. I. Quoted by Račić.
- NICAISE, *Soc. d. Chir.*, 1884. Quoted by Deeming.
- NICAISE, V., *Thèse de Paris*, 1904-5; *Arch. gén. d. Chir.*, 1909, iii, 453; *Encyclop. franç. d'Urol.*, 1914, iii, 75.
- NICOLICH, *Jour. d'Urol.*, 1912, ii, 843.
- NÚÑEZ, J. O., *Prog. d. l. Clin.*, 1935, xliii, 31.
- O'NEIL, R. F., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1929, xxii, 19 and 30.
- ORITZ, A. F., and GARCIA, J. E., *Rev. med. latin-amer.* 1933, xviii, 529.
- OULIÉ (4), *Bull. et Mém. Soc. nat. de Chir.*, 1928, liv, 718.
- * PARLAVECCHIO, *Zentralb. f. Chir.*, 1909, xxxvi, 1702.
- PEACOCK, T. B., and WALE HICKS, J., *Trans. Pathol. Soc. Lond.*, 1863-4, xv, 247.
- PELS-LEUSDEN, F., *Zentralb. f. Chir.*, 1931, lviii, 2984.
- PERESCHEVKIN. Quoted by Vedenski.
- PERRIN, *Lyon chir.*, 1929, xxvi, 707.
- PEYROT. Quoted by Nicaise.
- PITTS, A. G., *N.Z. Med. Jour.*, 1919, xviii, 191.
- PODDIGHE, A. (3), *Riforma med.*, 1929, i, 634.
- POESSLT, A., *Frankf. Zeits. f. Pathol.*, 1934, xlvii, 194.
- RAČIĆ, J. (10), *Zeits. f. urol. Chir.*, 1925, xvii, 163; *Zeits. f. Urol.*, 1931, xxv, 341; *Urologia*, 1934, i, 182; *Zeits. f. Urol.*, 1934, xxviii, 292.
- RAVASINI, C., *Atti d. Soc. ital. d. Urol.*, 1929, 130.
- * REGOLI, G., *Gazz. internaz. med.-chir.*, 1930, xxxviii, 293.
- RÉVÉSZ, *Zeits. f. Urol.*, 1926, xx, 66.
- ROCHE, E. H., and MACKENZIE, B., *N.Z. Med. Jour.*, 1935, xxxiv, 270.
- ROWLANDS, R. P., *Guy's Hosp. Rep.*, 1929, lxxix, 117.
- RUSSELL, J. I., and KILBANE, E. F., *Ann. of Surg.*, 1917, lxxv, 585.
- SABOLOTNOW. Quoted by Nicaise and Young and Davis.
- SALLERAS, J., *Rev. d. Especialid.*, 1928, iii, 402.
- SCHPEL, J. A. C., *Nederl. Tijds. v. Geneesk.*, 1930, lxxiv, 5705.
- SCHMIDT, L. E., *Trans. Amer. Assoc. Genito-urin. Surgeons*, 1915, x, 131.
- SENANTE, V. FERRANDIZ, *Rev. d. Cir. d. Barcelona*, 1931, i, 388.
- SIGINOLFI, P. (2), *Riv. radiol. e fis. med.*, 1930, Supp. No. 1, 129.
- SILNIKOV, S., *Nov. chir. arch.*, 1928, xvi, 563; *Zeits. f. urol. Chir.*, 1929, xxviii, 403.
- SORRENTINO, M., *Riforma med.*, 1930, xlvi, 783.
- SPURR, R., *Rev. d. Especialid.*, 1928, iii, 402.
- STARK, V. (2), *Vestnik khir.*, 1928, xiv, 92; *Zentralb. f. Chir.*, 1928, lv, 2888.
- STAVELEY, A. L., *Montreal Med. Jour.*, 1890, xviii, 148, quoted by Kretschmer.
- SURRACO, L. A., and associates (3), *Ann. de Fac. de Med.*, Montevideo, 1932, xvii, 668 and 818; *Rev. méd. latin-amer.* 1931, xvi, 1453.
- TARNOWSKY, G. DE, *Surg. Clin. N. Amer.*, 1927, vii, 783.
- TAUBENSCHLAG, H., *Bol. y Trab. Soc. de Cir.*, 1925, ix, No. 4.
- THOMAS, D., *Lancet*, 1879, i, 297; *Austral. Med. Jour.*, 1881, ii, 250, 344, 391, and 489; *Proc. Roy. Soc.*, London, 1885, xxxviii, 457.
- TOOLE (46 renal, 10 pararenal), *Arch. f. klin. Chir.*, 1930, clix, 124.
- VALDONI, P. (10), *Clin. chir.*, 1928, iv, 981.
- VASILIU, SILVESTROVICI, and FRANKE, O., *Rev. san. mil.* 1934, xxxiii, 371 and 474.
- VEDENSKI, D. A. (6), *Vestnik khir.*, 1928, xiii, 129; *Zeits. f. Urol.*, 1925, xxii, 694.
- VILA, E. L. (8), *Rev. d. Cir. B. Aires*, 1929, viii, 158; *Bol. y Trab. Soc. de Cir.*, 1928, xii, 785.
- VON DER BECKE, A., *Rev. d. Especialid.*, 1928, iii, 402; *Rev. med. int. y tis.*, 1927, iii, 458; *Semana méd.*, 1927, ii, 1186; *Rev. d. Especialid.*, 1927, ii, 1029.
- WEDENSKI, D. A., see Vedenski.
- WEINBERG, M., *Ann. de l'Inst. Pasteur*, 1909, xxiii, 472.
- WELSH, D. A., and CHAPMAN, H. G., *Lancet*, 1908, i, 1338; 1909, i, 1103.
- WILLANS, E. T., and STEWART, M. J., *Brit. Jour. Surg.*, 1927, xiv, 667.
- WOODMAN, G. S. (2), *Brit. Jour. Urol.*, 1931, iii, 299.
- YOUNG, E. L. (3), *Cabot's Modern Urology*, 1924, 2nd ed., ii, 519. New York.
- YOUNG, H. H. and DAVIS, D. M., *Young's Practice of Urology*, 1927, i, 358.

CONGENITAL DISLOCATION OF THE HEAD OF THE RADIUS

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THERE is very little if any reference in the British literature to congenital dislocation of the head of the radius. It is barely mentioned in the surgical text-books and then only as part of another condition. Dislocation associated with congenital synostosis of the upper end of the radius and ulna is not a true dislocation, for the head is fixed by the fusion just below it, and may lie posterior to the ulna or anterior, or may in fact be non-existent. In some cases of birth palsy (Erb's type) a subluxation of the head of the radius develops as a result of muscle unbalance.

In the condition I am describing there is no fusion of the radius and ulna and no paralysis. The head of the radius is displaced forwards, is quite free from the ulna, and can be moved through a considerable range, but cannot be replaced in the normal position. This is significant because traumatic dislocation of the head of the radius, particularly if uncomplicated by fracture of the ulna, is reduced with ease and often spontaneously.

My attention was first attracted to the condition by being asked to treat a child who, having recently fallen, had apparently dislocated the head of his radius, and a house surgeon had twice unsuccessfully attempted reduction. I also failed by closed manipulation, and at open operation I found that the head of the radius was outside the capsule; that there was no orbicular ligament; that there was no lesser coronoid fossa; that the head of the radius was misshapen; and that the radius was too long, so that when one pushed the head against the capitellum, pressure was very great. I accordingly removed the head of the radius. I was able later to compare it with a dislocated head removed for mal-union of the ulna. The congenital head is small. Instead of being flattened as shown in *Fig. 17*, it is almost domed as in *Fig. 18*, with only a little dimple in place of the broad shallow capitellar depression (see *Fig. 29*).



FIG. 17



FIG. 18

About a year later another child was presented with the same story of injury. I regret to say that I did not recognize the significance of failure to obtain reduction by closed manipulation until I had again opened the elbow-joint. It was then immediately apparent to me that I was dealing with the same condition, and that it ought to be possible before operation to establish the congenital nature of the dislocation.

In radiographs the radius appears too long for the ulna, and the head of the radius appears ill-formed. There is a small area of ossification in the tissue overlying the head of the radius resembling the sesamoid type of calcification which sometimes occurs over the head of a congenitally dislocated femur. Subsequent cases showed that this 'sesamoid' was not constant. There is an anterior curve

of the posterior outline of the ulna, starting at the level of the coronoid fossa as in *Fig. 19*. It is sometimes slight but definitely contrary to the slightly posterior curve normally present at this level* (*Fig. 20*). A variation is also noticeable in



FIG. 19



FIG. 20



FIG. 21



FIG. 22

the anterior outline of the ulna. Instead of the backward sweep from the coronoid (*Fig. 22*) there is almost a straight line (*Fig. 21*). These features, together



FIG. 23.—Case 1. Congenital dislocation of the head of the radius. Note: (1) The radius is long; (2) The head is poorly formed; (3) The posterior outline of the ulna has a forward curve starting high up; (4) The anterior outline of the ulna has not the usual backward curve from the coronoid. (Cf. *Fig. 25*.)



FIG. 24.—Case 1. Congenital dislocation of the head of the radius. Six months after replacement of the head of the radius. The X-ray shows pressure atrophy and partial re-dislocation of the head. The shape of the upper end of the ulna is of course unchanged from that shown in *Fig. 23*.

* There is normally a faint anterior curve starting about the junction of the upper and middle thirds.

with the inability to reduce the head, point to a congenital dislocation rather than an acquired one.

When some time later a third case presented itself, it was easy to recognize



FIG. 25.—Case 2. Congenital dislocation of the head of the radius.



FIG. 26.—Case 3. Congenital dislocation of the head of the radius. Note the long radius and poor head and the shape of the ulna. See Fig. 27 for photograph of arm and Fig. 29 for scale drawing of head after removal.

the nature of the condition (Fig. 23). At operation the diagnosis was confirmed and on the suggestion of another surgeon I replaced the head of the radius. I did this against my inclination because I believed that a pressure atrophy (such as I



FIG. 27.—Case 3. Photograph of congenital dislocation of the head of the radius. Note posterior curve in outline of ulna. See Fig. 26 for X-ray, and Fig. 29 for scale drawing of head of radius after excision.



FIG. 28.—Case 3. Photograph of normal elbow. (Cf. Fig. 27.)

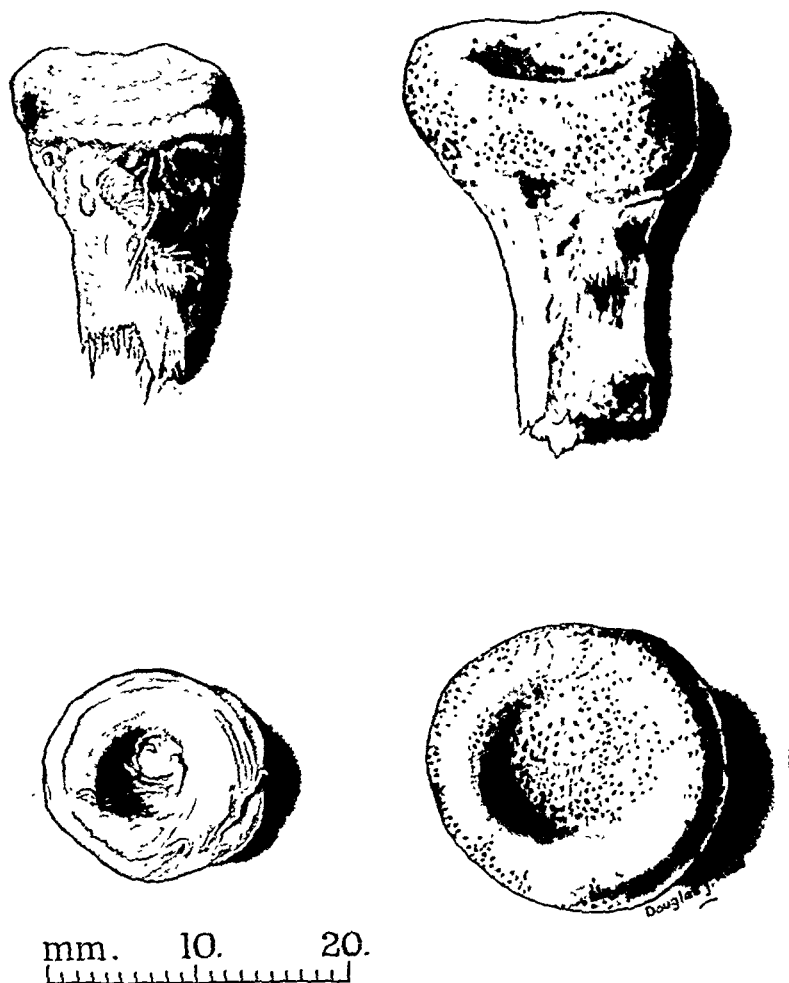


FIG. 29.—Scale drawings of dislocated heads of radius from patients of similar ages. On the left is the congenital type removed from Case 3 (see Figs. 26, 27). On the right is a normal head dislocated in association with mal-union of the ulna. (See Fig. 30, Case 4.)

have described in the head of the femur when an old-standing congenital dislocation is reduced) would occur and stiffness would result. This was so, and later I had to remove the head of the radius. (*Fig. 24.*)

It must not be assumed that it is always necessary to operate. Only for such patients as have loss of function should operation be undertaken.

Eleven children have been treated. In each case the dislocation was anterior. In none was the condition bilateral. Only five needed operation. (*Figs. 23-37.*)

Children usually attended following injury previous to which misplacement had been unnoticed. On clinical examination the head of the radius was felt to be prominent in the antecubital fossa near the mid-line and irreplaceable. There was, of course, a hollow where the head of the radius should be, and the slight anterior ulnar curve noticeable in the radiograph was clinically appreciable (*see Figs. 27, 33*). In over half the cases function was unimpaired. In no instance was



FIG 30—*Case 4* Traumatic dislocation of the head of the radius associated with mal-union of the ulna. *See Fig. 29* for scale drawing of head after removal. Compare shape of head with *Figs. 23, 26*, etc. Also note normal curves of ulna.

extension or rotation affected. Five patients suffered from continual or intermittent blocking of flexion. In such cases it is a simple matter to excise the head of the radius. For reasons I have given I am opposed to attempting to replace the head. For successful reduction the child must be young, preliminary traction must be employed, and at operation an orbicular ligament must be formed.

The X-ray appearances I have described are shown in *Figs. 23-26, 31, 32, 36, 37.*

Congenital dislocation has an added importance in that a doctor may be blamed for not having efficiently treated an arm, the dislocation having been unnoticed before the injury for which treatment was started. *Fig. 35* brings out this aspect. The boy had sustained a fracture of the ulna for which he had been treated by a general practitioner. His parents complained of: (1) A lump on the back of the arm; (2) The lump in front of the elbow interfering with function. It was seen from the radiograph that the alinement of the ulna was adequate and union was satisfactory. I was able to establish that the head of this radius had been dislocated before the accident, and was in fact a congenital dislocation. The lump on the back of the arm was of course merely callus and gradually disappeared.



FIG. 31.—Case 5. The upper radiographs show the dislocated right joint. Those below, showing the normal left elbow, have been reversed in printing to make comparison easier. Note the changes in the radius and ulna and especially the excessive length of the radius.

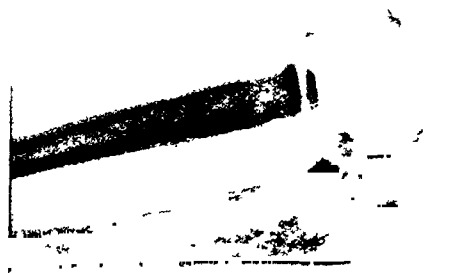


FIG. 32.—*Case 6.* Congenital dislocation of the head of the radius. Note the ossification in the capsule. (Cf. *Figs. 33, 34.*)



FIG. 33.—*Case 6.* Photograph of congenital dislocation of the head of the radius. Note the anterior curve of the posterior outline of the ulna. (Cf. *Fig. 34.* See *Fig. 32* for radiograph.)



FIG. 34.—*Case 6.* Photograph of normal elbow. (Cf. *Fig. 33.*)



FIG. 35.—Case 7. Fractured ulna and congenital dislocation of the head of the radius. This diagnosis may seem unjustified, but Fig. 36 shows an elbow of a child nearly the same age in which the dislocation was traumatic and associated with fracture of the ulna. That the changes typical of congenital dislocation are present in the ulna of Fig. 35 and not in Fig. 36 is made more clear in Fig. 37.

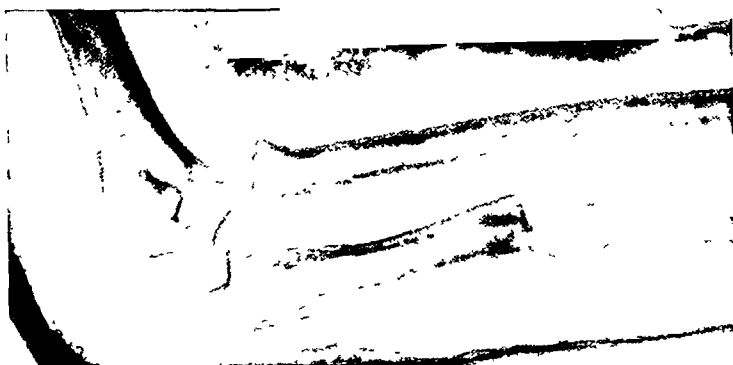


FIG. 36.—Case 8. Traumatic dislocation associated with fracture of ulna. Age only slightly less than Case 7. (Cf. Fig. 37.)

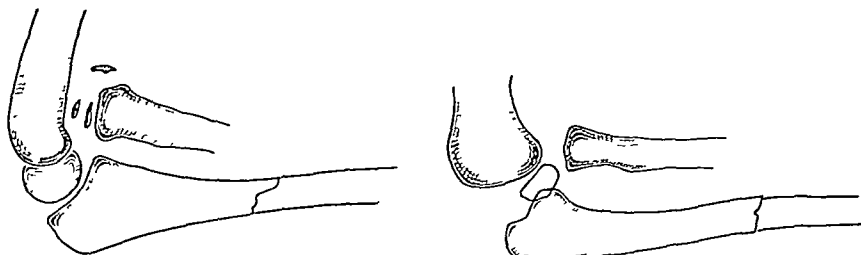


FIG. 37.—Cases 7 and 8 contrasted. The deformity of the ulna due to fracture has been discounted by tracing the radiograph so as to reconstruct the ulna.

SUMMARY

A congenital dislocation of the head of the radius is described, setting out the clinical, radiological, and operative observations on eleven cases. The dislocation was in each instance anterior and unilateral. In only five patients was function interfered with and operation undertaken. Removal of the head is preferable to replacement except under certain specified conditions.

MULTIPLE PRIMARY EPITHELIOMA IN LYMPHATIC LEUKÆMIA

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MULTIPLE primary carcinoma of the skin has often been described. It occurs in X-ray workers and in xeroderma pigmentosum, and is particularly associated with those whose work, as in the case of sailors, leads to constant exposure to the elements. In the following case the occupation of the patient, a grave-digger, brings him into this category. The case is, however, unusual in that his cutaneous condition was in part carcinomatous, and in part a complication of lymphatic leukæmia.

The patient, a grave-digger, aged 66, was admitted to Westminster Hospital in March, 1929, with a three months' history of ulcer of the lower lip. A portion



FIG. 38.—Section of tumour of lip, March, 1929. Squamous-cell carcinoma.

was removed, and showed a typical well-keratinized squamous-cell carcinoma (*Fig. 38*). The corium was densely infiltrated with lymphocytes. There were a few submental glands. He received 936·8 mg.-hr. radium to the lesion, and 1685·6 mg.-hr. to glands. In August, 1929, the original lesion was apparently cured; there were some palpable cervical glands, and these received 3007·6 mg.-hr. of radium. They returned practically to normal size.

No blood examination was performed at the patient's first admission, and the tumour of the lip was diagnosed as a squamous-cell carcinoma without qualification. Later, however, it was re-examined in the light of the multiple lesions, and the histology appears identical with them. The amount of lymphocytic infiltration of the corium was very much greater than is normally found in squamous-cell carcinoma, although, of course, such infiltration is almost always present to some extent.

On admission in August, 1933, the patient stated that he had remained well until April of that year, when a small nodule appeared in the skin of the right malar region. It became larger and broke down in two months; it never healed. Following this lesion many others of similar nature appeared elsewhere, on hands



FIG 39 —Appearance of face, August, 1933

and face, and followed the same course (*Figs. 39, 40*). Œdema of the face followed the appearance of the malar lesion. He complained of nothing else.

He was seen by Dr. S. E. Dore, who reported, "Lesions on face consistent with those sometimes found in lymphatic leukæmia, but the ulcers resemble epitheliomata. The condition of the skin on the back of the hands is like that of 'sailor skin' produced by exposure. The recent tumour formation (on the hands) resembles that of the face."

The original lesion of the lower lip now showed soft and painless skin of scar-tissue type. On the right side of the face were numerous ulcers with raised and thickened edges, and depressed central pits showing no attempt at granulation. There was marked œdema of the right side of the face; the skin was pendulous

over the mandibular area. There were many enlarged lymph glands in the right cervical chain; the glands on the left side, previously treated by radium, had not increased in size. The superficial lymph glands all over the body were readily palpable. There was a large scrotal hernia. Neither liver nor spleen was enlarged. The hands and arms showed lesions similar to those on the face. The rest of the skin was normal; there were no purpuric lesions and the patient did not complain of pruritus. There were numerous retinal hæmorrhages. There were no other abnormal physical signs. Blood examination showed: R.B.C. 1,910,000, Hb 23 per cent, C.I. 0.6, W.B.C., 226,400. All the cells seen were primitive lymphocytes, with prominent nucleoli. The Wassermann reaction was negative, and the urine normal.



FIG 40—Appearance of arms and hands, August, 1933.

A biopsy was performed by Mr. E. Rock Carling, and showed the following results. (Figs. 41, 42.)

Axillary lymph gland. Much enlarged. The germinal centres are not visible, and the general structure altered. It consists exclusively of small deeply-staining cells of the general appearance of lymphocytes. There is no evidence of squamous-cell carcinoma, and the picture is typically that of lymphatic leukæmia.

Small non-ulcerated lesion of back of hand. Pronounced œdema of cutis vera. The epithelium is much thinned, the papillæ are much prolonged, and there is diffuse lymphocytic infiltration. The whole picture is that of psoriasis.

Two lesions of hand, and large ulcer of face, central area. Typical well-keratinized squamous-cell carcinoma, penetrating deeply. There is a massive infiltration with lymphocytes, but this is frequently found in normal squamous-cell carcinoma.

Large ulcer of face, periphery. The epithelium is very thin, and there is a dense infiltration with lymphocytes extending throughout the corium and infiltrating

the fat. The papillæ are absent as in scar tissue. There is also an area of early squamous-cell carcinoma.

The patient left the hospital and died at home ; there was no autopsy.



FIG. 41.—Section of tumour of face, August, 1933. Leukæmic deposit in skin with early squamous-cell carcinoma

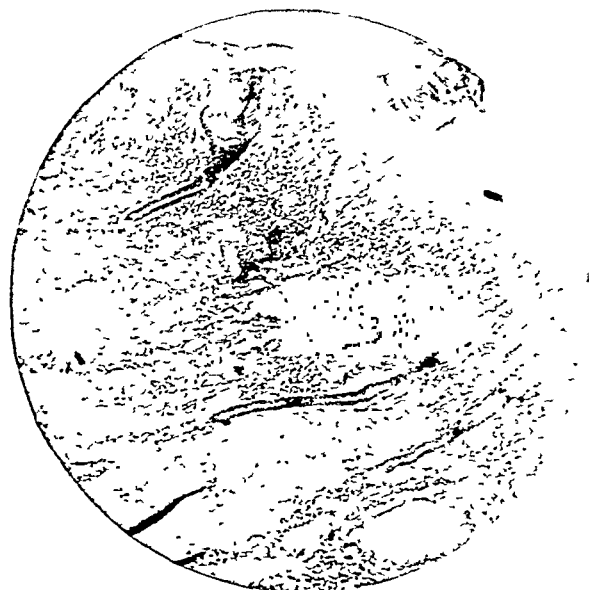


FIG 42.—Section of lesion of hand, August, 1933. Thinning of epithelium ; downward processes elongated.

DISCUSSION

Lymphatic leukæmia is usually considered to be a neoplastic disorder, and the association of two distinct malignant conditions, while of interest, is familiar enough. The chief point of note in this case is its bearing on the function of the lymphocyte in malignancy. Murphy has published much work on this subject, starting from the observation that there is consistently a lymphocytic infiltration at the growing edge of a malignant neoplasm. He argued that the lymphocyte provided an important barrier to the spread of tumours.

There is, however, evidence leading in the opposite direction. In cutaneous epithelioma invasion only occurs into tissues where lymphocytes are aggregated; and lymphatic glands are of all organs the most prone to secondary invasion.

Leukæmic deposits in the skin are of frequent occurrence, and take many forms. The condition is fully described by Hirschfeld. In a long series of references no allusion is made to the development of malignant changes in the superficial epithelium, and it has not been possible to find any case of such a change. It seems probable, therefore, that the primary exciting factor for the epitheliomata was the long-standing condition of xeroderma. In every case, however, the epitheliomata in fact developed in areas of leukæmic infiltration, and this suggests that nutritional or other changes occasioned by their presence were, for the already pathological epithelium, the last straw. It is certainly remarkable that epitheliomata consistently develop in areas affected by injuries of long standing, where subepithelial fibrosis is a feature; we find this in lupus, old burns and scars, and syphilis. There may be a purely nutritional basis for this fact; and the leukæmic infiltration may have precipitated in this case malignancy in which a defective blood-supply to the epithelium was one factor.

My thanks are due to Mr. E. Rock Carling for permission to publish this case, and to Dr. R. G. Waller for the photographs.

REFERENCES

-
- HADFIELD, G., and GARROD, L. P., *Recent Advances in Pathology*, 1932, 84. London.
HIRSCHFELD, H., *Krankheiten des Blutes*, 1925, i, Berlin.

ACUTE ILIO-PSOAS ABSCESS

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IN recent years suppurative conditions within the sheath of the ilio-psoas muscle have usually been assumed to be tuberculous and derived from some part of the vertebral column or from an associated joint. There is, however, a subacute or acute lesion, characterized by the formation of a large abscess, from which pyogenic organisms may be readily obtained. The origin of such an abscess may be from any one of a variety of sources; it may be a blood- or lymph-borne infection, or may be the result of a direct extension through the sheath from a neighbouring associated structure. The case described below belongs to the last group, being the result of a perforation of the cæcum, and in one or two of its features would seem to be unique.

HISTORY.—The patient was a man of 62 years, who for three months had complained of vague central abdominal pain. There had been general malaise and some loss of weight, with marked flatulence. He had not noticed nausea or sickness, and the bowel had been opened twice or three times daily during several previous weeks, the stool being loose, but otherwise normal. During this period he attended as an out-patient, and a radiological examination of the gastrointestinal tract was carried out. No abnormality was detected.

Two or three days before his admission to hospital, the pain had become more severe, all over the right side of his abdomen, from the costal margin to the groin, being most severe in the lower quadrant. He had not vomited, though diarrhoea had supervened; micturition was normal.

ON EXAMINATION.—The patient's temperature was 99·8° and his pulse 88, with respirations 23 per minute. He was a thin, ill-nourished man, with muddy complexion and moist, slightly furred tongue. The abdomen, poorly covered, showed diminished respiratory movement and tenderness over the whole of the right side, increased below the umbilicus and reaching a maximum in the groin. The tonus of the abdominal musculature was increased a little in the right lower quadrant, though true rigidity was absent. There was no palpable swelling. Rectal examination showed no abnormality, and the urine was clear and normal. The right thigh was held in slight flexion and external rotation at the hip-joint, the contour being normal.

The patient was placed under observation, and during the succeeding twenty-four hours his pulse and temperature remained steady. The pain in his abdomen diminished in the upper part, but increased in the groin, the increased tonus persisting, but in lesser degree. The thigh became more flexed and rotated outwards and some swelling became evident. This was at first seen as a fullness in Scarpa's triangle, spreading gradually to the inner and posterior aspects of the limb. Attempts to extend the thigh and rotate it inwards were strongly resisted by the patient, though further flexion was not resented.

Forty-eight hours after his admission, the abdominal signs had largely disappeared, and the focus of his complaint seemed to have moved to his thigh. There was still no nausea or sickness, though the diarrhoea persisted, the stools being free from blood. The thigh had become increasingly swollen, and œdema appeared over Scarpa's triangle and over the adductor area. The limb lay heavily on its outer side, now flexed to fully a right angle at the hip. Percussion upon the greater trochanter failed to elicit pain, and there was no clinical evidence of lesion in either the spine or bony pelvis. This was confirmed by radiological examination. An X-ray photograph of the right hip-joint, however, showed a collection of gas around the neck of the right femur (*Fig. 43*), and it was concluded that an appendix abscess



FIG. 43.—X-ray photograph of the right hip-joint, showing a collection of gas around the lesser trochanter and neck of the femur. The abduction and external rotation at the joint are evident.

was presenting in the thigh, having passed along the psoas sheath. Neither swelling nor fullness was palpable above the inguinal ligament.

OPERATION.—The general condition of the patient was very poor; the pulse was of low tension and had risen a little, and it was felt that he would successfully withstand only the minimum of surgical interference. Accordingly, under gas and oxygen anaesthesia, an aspirating needle was passed into the swelling in Scarpa's triangle, to the outer side of the femoral vessels. At once gas with faecal odour escaped, and thin sanious pus was aspirated. With the needle still in position, a free vertical incision was made into the abscess cavity; this was extensive, passing

beneath the femoral vessels, down to the neck of the femur and between adjacent muscles. The wound was left widely open, being lightly packed with gauze. Bacteriological examination of the pus showed coliform bacilli and streptococci, and a profuse growth of these organisms was obtained.

During the next few days the general state of the patient deteriorated further. A profuse discharge occurred from the wound, purulent at first, faecal later. He died on the tenth day after his operation.

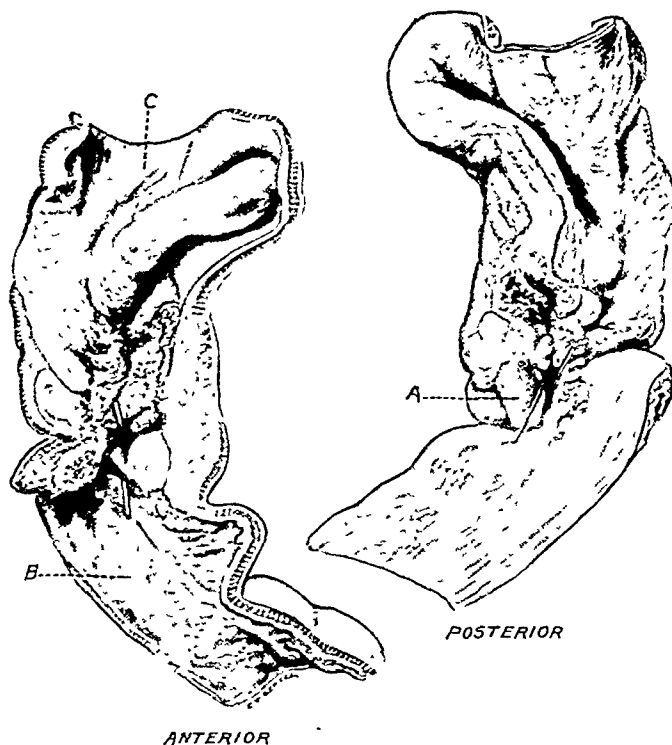


FIG. 44.—Carcinoma of cæcum, enveloping the appendix, and showing two perforations. A, Appendix; B, Ileum; C, Ascending colon. ($\times 2$.)

AUTOPSY.—A small carcinoma was found in the cæcum (*Fig. 44*). It was placed upon the posterior wall of the bowel and infiltrated the lateral walls, projecting a little above the surface of the mucosa. Its base showed ulceration and a small double perforation. The appendix was almost completely enveloped in the neoplasm, its tip being the only recognizable part. A large abscess cavity was situated behind the cæcum, extending outwards for a little way across the iliacus, and downwards to the thigh, within the psoas sheath. The psoas was represented by a dark confluent mass, with pus around and within it, and led downwards beneath Poupart's ligament to a large excavated space in the upper part of the thigh. This

cavity extended on each side of and beneath the femoral vessels, and led down to the medial side of the femoral neck. The vessels and nerves stood across it, cleanly dissected, and the space could be traced among the muscles around it. It contained a brownish, odorous pus.

Microscopic section of the tumour showed a columnar-celled carcinoma, with alveolar formation and with abundant fibrosis. Its origin, whether from the cæcum or proximal part of the appendix, is uncertain.

DISCUSSION

Acute suppuration within the psoas sheath has been very fully described, especially during the latter half of the last century, and in recent years a considerable number of cases have been recorded, though reference to it is uniformly omitted from our surgical text-books. In 1878 Margano¹ produced a monograph dealing with acute psoas infection following trauma. Many examples are cited therein, with abundant evidence of the source of the abscess. The author traces references to records which appeared many years before his time. Apparently the first description was made by Fabrice de Hilden,² who, in a letter in 1682, tells of a young man who tripped and fell backwards, bruising his back. There followed an abscess in his psoas muscle which was opened by his surgeon. In 1797 Déramé described fully a similar case, proved at autopsy.

Acute psoas abscess of intestinal origin has been recognized for many years, and among the earlier references are those of Ménière in 1828, and Dance in 1832. Since that time many more examples have been recorded, but they all refer to typhlitis or appendicitis as the forerunner, and thus occurred on the right side.

Direct spread of pus from a perinephric abscess, from parametritis, from an empyema thoracis, or from acute osteomyelitis of the ilium, has led to the formation of a massive abscess in the thigh. A psoitis arising in the course of a general pyæmia has in more recent times been fully described, and cutaneous suppuration and nasopharyngeal infection have supplied the primary focus, with the staphylococcus and streptococcus predominating as the causative organisms. The condition is not uncommon in children, and has been described by Ingelrans and Minne,³ Behrman,⁴ and Sworn,⁵ within recent years.

The close anatomical relationship to the inguinal, iliac, and lumbar lymphatic glands has brought upon the psoas muscle infections derived from the lymphatic areas drained by those systems, particularly from the lower limb. The lumbar retroperitoneal lymph nodes have been especially blamed by Rogers,⁶ though his evidence is not very convincing.

Pus may be localized to various parts of the psoas sheath, and occasionally it spreads out across the iliacus muscle. Typically, the fully developed abscess presents a deep and tender swelling above the inguinal ligament connected to a second below that structure. Passing out of the sheath at the neck of the femur, an extensive suppuration often burrows among the flexor and adductor muscles of the thigh, and so may pass on down the limb. On opening such an abscess below the inguinal ligament, there is displayed a cavity which may be widespread among the muscles, across which the femoral vessels may stretch unsupported, and in which the femoral nerve, dividing into its many branches, may stand out cleanly dissected. As in other forms of psoas abscess, pus may

reach to a point as distant as the heel before presenting. An interesting record is made by Layton,⁷ who described an example in which, on account of long recumbency, the reverse process occurred. A popliteal adenitis derived from erysipelas of the heel resulted in an abscess which tracked upwards through the thigh to Scarpa's triangle, there entered the psoas sheath, and ascended through the abdomen to the thorax.

The flexed and externally rotated thigh is characteristic and increases as the condition advances. It has been said that "where the massive thigh lies flexed and immovable upon its outer side, a fatal termination is to be expected, however free the drainage that be instituted."

I am indebted to Mr. F. D. Saner for kindly giving me permission to publish this case.

REFERENCES

- ¹ MARGANO, G., "La Psoïte traumatique", *Trans. Roy. Med. and Chir. Soc. Paris*, 1878.
- ² FABRICIUS HILDANUS, *Observationum et Epistolarum Chirurgico-mediarum*, 1716, i.
- ³ INGELRANS, P., and MINNE, J., "La Psoïtis de l'Enfant et de l'Adolescent", *Arch. franco-belges de Chir.*, 1930, xxii, Dec., 1930.
- ⁴ BEHRMAN, S., "Non-spinous Ilio-psoas Abscess", *Lancet*, 1930, ii, 297.
- ⁵ SWORN, B. R., "Acute Psoas Abscess", *Brit. Med. Jour.*, 1933, ii, 6.
- ⁶ ROGERS, M. H., "Psoas Abscess from Lumbar Retro-peritoneal Lymph Glands", *Amer. Jour. Orthop. Surg.*, 1911, ix, 232.
- ⁷ LAYTON, T. B., "An Unusual Case of Psoas Abscess", *Guy's Hosp. Gaz.*, 1912, xxvi.

CLEFT STERNUM: CASE REPORT AND BRIEF COMMENTARY

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COMPLETE median cleft or fissure of the sternum has been described by various observers, usually associated with defects of the neck and abdominal wall. The authors feel that the case to be described here, having no detectable abnormality other than the sternal deficiency and a superficial scar of the abdominal wall, is therefore worthy of record.

HISTORICAL SURVEY

Gibson and Malet¹ (1879) described a case in which a sternal fissure existed in the cranial end of the bone as far downwards as the upper border of the fourth rib; this was associated with a strong membrane attaching the sternal edge in the cleft to the cricoid cartilage, and a scar-like strip of skin, 3 in. long, passing upwards from the umbilicus. Professor Sir William Turner² (1879) described a specimen in the Anatomical Museum of the University of Edinburgh, with fissure of the sternum ending at the level of the seventh rib, where the two halves articulated in the mid-line. He quoted cases of Herr Groux, Skoda and Forster, the former describing, apparently, his own condition, and the latter found the ribs to be fused to the sternum without articulation.

Martin³ (1887) described a case in a new-born female, which he traced to be healthy to the age of $4\frac{1}{2}$ months, though with umbilical hernia, divarication of the rectus muscles, and attenuation of the skin of the precordial area.

More recently Barillet⁴ described a case in which the cleft was sufficiently large to permit examination and direct record of the aortic pulse. Benhamou, Hermann, and Levi-Valensi⁵ have described a case of apparent complete absence, as opposed to fissure, of the sternum, with records of cardiograms obtained directly through skin from the pericardial surface of the heart as far out as the apex. It is interesting to note that no cardiac abnormality could be detected.

The condition was exhaustively reviewed in 1922 by Alfred Szenes⁶ and in 1926 by Mr. David Greig, of Edinburgh.⁷ Szene's case was a male aged 8 years, with complete fissure of the manubrium and body of the sternum as far as the xiphoid at the sixth costal cartilages. Scarred skin lined the fissure and a ligamentous raphe connected a spur-like protuberance of the chin with the sternal fissure, over which it spread fan-wise. This raphe caused a pouch-like fullness of the neck and limited extension of the head on the neck.

Morton and Jordan⁹ (1935) described a still further stage in a case of median cleft of the lower lip and mandible, cleft sternum, and absence of the basi-hyoid. In their case the sternal deformity consisted of apparent absence of the manubrium and upper half of the sternum, with the inferior gnathoschisis and the connecting area of scar tissue from the mental region to the floor of the sternal cleft.

CASE REPORT

A male Arab, aged about 20 years, weaver, of Shaikh Othmann, Aden, was seen by Dr. Napier, of the Keith Falconer Hospital, in November, 1935, on account of ulcers of his legs. In the course of examination Dr. Napier discovered the condition to be described, and very kindly brought it to our notice.

The condition—apparent absence of the sternum—had been present since birth; there were no relative symptoms. The patient and his parents were, in fact, surprised that it should evoke more than passing interest. Family history, as far as could be ascertained, revealed no other case with congenital abnormality of any kind.

ON EXAMINATION.—The patient was somewhat below the normal stature for his age, and of a poor type mentally; the general development was otherwise a fair average of the local type of Arab and he followed his employment regularly. Ulcers were present on the lower halves of both legs, not associated with varicosities or œdema of the feet, and appeared to be of the tropical type which is common amongst the natives of this district.

On the anterior aspect of the thorax in the mid-line, the area normally occupied by the sternum appeared to be replaced by a well-marked vertical cleft (*Figs. 45, 46*) which is described

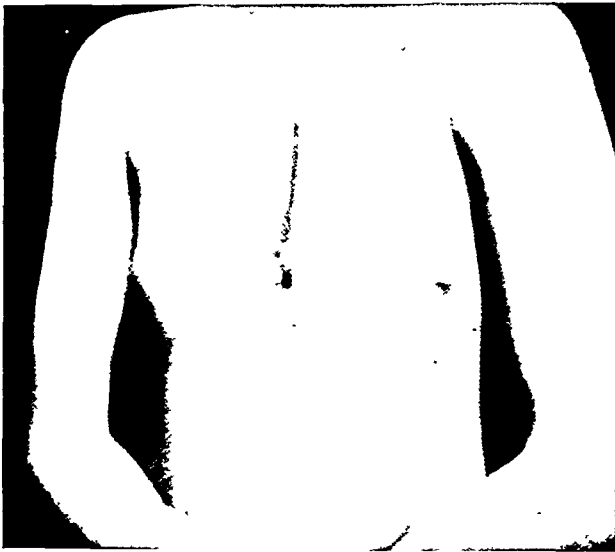


FIG. 45.—Photograph showing condition of sternum.

in detail below. There was, in the floor of this depressed area, opposite the fourth costal cartilages, a circular, puckered scar, $\frac{1}{2}$ in. in diameter, soundly healed and not adherent to deep structures.

In the mid-line of the abdomen was a slightly raised linear scar, 2 in. long, ending below at the umbilicus. Both scars had been present since birth. There was no history of trauma. The recti abdominis muscles were normal, there was no herniation at the umbilicus, and the symphysis pubis was normal.

The mouth, tongue, and mandible were normal, as was also the neck. A degree of deficiency of pubo-axillary hair was noted, but otherwise the pudenda were normal. No other abnormality could be detected on examination.

THE STERNUM.—The manubrium and body of the sternum were found to be deficient in the mid-line as far, inferiorly, as the xiphisternum; laterally, remains of these portions were present, so that the manubrium and body of the sternum were, in effect, split into two portions by sagittal section. Integument passed into the cleft so formed, and, except for the puckered scar noted as being opposite the fourth costal cartilages, was of normal texture. A regular pulsation was visible in the lower third of the cleft.

Mensuration.—In the resting decubitus position the space between the inner sternal edges at the upper end of the cleft was $1\frac{1}{2}$ in.; during full inspiration this increased to $1\frac{3}{4}$ in. and it decreased to $1\frac{1}{4}$ in. during full expiration. In the sitting position, at rest, this measurement was 1 in. At the nipple line, in resting decubitus, the space was $1\frac{1}{2}$ in. wide, whilst at the lower end, opposite the sixth costal cartilages, the space measured 1 in.; the latter measurement did not vary with position or with respiration.

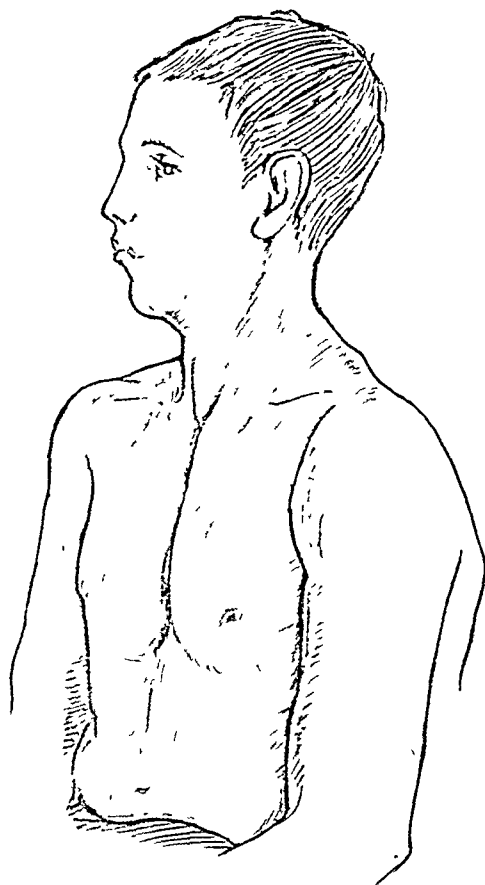


FIG. 46.—Another view of the condition. (From a pencil sketch by Mrs. Haythornthwaite.)

Anatomy.—

The borders of the cleft.—

Superior: No border could be defined and the cleft passed without interruption into an apparently normal neck.

Inferior: A definite, bony-hard margin, concave superiorly, at the level of the sixth costal cartilages; this appeared to be firmly attached to the lateral borders of the cleft, and on palpation resembled the upper border of a small ill-defined xiphisternum, the total length of which was not more than $\frac{1}{2}$ in.

Lateral: Smooth osseous edges, $\frac{1}{4}$ in. thick, extended on both sides the full length of the cleft; to the lateral margins of these osseous plates, which were about $\frac{1}{2}$ in. wide, were attached, in normal disposition, the clavicles and costal cartilages.

The floor of the cleft.—The depth of the floor, below the level of the adjacent chest wall, was seen to vary with respiration: in the resting position one finger could be laid in the cleft and was then flush with the edges. In expiration a degree of shallowing occurred, whilst during the act of coughing the floor rose above the level of the surrounding edges so that considerable herniation of the pleura or mediastinum occurred.

The deep relations of the floor, from above downwards, were: Trachea, deeply placed and covered by soft connective tissue. Just below the level of the second costal cartilages a definite fibrous band could be felt crossing transversely; this appeared to be the upper border of a thickening of fibrous tissue some 1 in. wide from above downwards, the lower border being less definite and apparently merging into the pericardium. From this lower border to the upper edge of the xiphisternum the floor consisted of pericardium covering heart and great vessels, the determination of the exact anatomy of these being indefinite. The pulsations were, however, obvious on first looking at the patient, and, on palpation, the contractions could be appreciated just under the fingers: deeper palpation in this area was not tolerated by the patient.

THE CHEST.—Circumference at the nipple line, resting, was 33 in. The full respiratory excursion was only $\frac{3}{4}$ in. Clinical examination revealed no abnormality in the pulmonary or in the cardiovascular system. These findings were confirmed by X rays, the latter showing also the outline of the cleft sternum until in the lower half it is obscured by the cardiac shadow.

ALIMENTARY SYSTEM.—Mouth normal; the position and function of the œsophagus, stomach, and duodenum were examined by a small barium meal and observed by screening to be normal.

The Meinicke reaction was strongly positive; no other abnormality could be discovered in the examination of the patient.

COMMENTARY

Development.—The sternum develops, according to Keith,⁸ as a condensation of mesoderm on each side of the mid-ventral line, then represented by the primitive linea alba, at the fifth week. These form the right and left mesenchymal sternal bars, which are continuous anteriorly with the bases of the ventral part of the shoulder girdle, and are situated over the pericardium between the mandible in front and the umbilicus behind. Fusion of these sternal bars in the mid-line commences at the presternum about the 13-mm. stage (end of sixth week); the caudal portions are kept apart by the protrusion of the heart and liver until union takes place by the 34-mm. stage (tenth week). Chondrification and ossification then proceed.

Defects.—Bifid xiphisternum is common, whilst occasionally a median foramen is found in the region of the lower sternal segments. Fissure of the manubrium, extending a variable distance into the body, would appear to be a more usual occurrence than complete fissure of the sternum. Deficiency of the caudal end only is found in association with a gross embryonic lesion such, for example, as ectopia cordis.

Causative Theories.—Szenes,⁶ reviewing the point, favours some cause of obstruction operating between the cranial ends of the sternal bars, about the 15-mm. stage, relieved at a later stage to allow union of the caudal portions. He puts forward, as this cause, pressure, probably due to deficiency of amniotic fluid, and adhesion between the chin and chest wall during the stage of acute flexion, relieved later by extension of the head. In support he quotes a case of von Mall of an anencephalic embryo with the face adherent to the chest wall. Morton and Jordan⁸ appear to have arrived at a similar conclusion, and stress the importance of transient adhesions rather than pressure.

The case described in this paper would appear to be one of complete sternal fissure with fewer complicating features than we have been enabled to trace in other cases published. The scarring described may be regarded as a result of those influences which in other cases have produced gross lesions, and in this case failed to do so. The influence of some external or ectodermal (amniotic) factor seems to be supported, and the differentiation of these cases from defects of inclusion of viscera, such as ectopia cordis, made clear by the fact that the defect is usually in the cranial end of the sternum, and not in the caudal end as might be expected in these cases on embryological grounds. It is the caudal end which Keith⁹ and others maintain is last to unite, on account of the protrusion of the large heart and liver.

The time at which this defect originates may be supported as that at which the chin, chest wall, and umbilicus are closely apposed, synchronous with union of the cranial ends of the sternal bars, and separated in time to permit the caudal ends to come together, i.e., between the 13-mm. and 30-mm. stages.

Clinically, interest lies in the fact that no symptoms arose from this condition and that interference was not indicated for the complete sternal defect *per se*. The heart was not embarrassed, and we suggest that the thickened fibrous fold described in the floor of the cleft is a strengthened superior sterno-pericardial suspensory ligament which is described as arising normally from the dorsal surface of the angle of Ludwig.

In conclusion we wish to thank Dr. Napier for having shown us this case and inviting us to carry out investigations; and to express our sincere gratitude to Professor Grey Turner, who, in a short visit to Aden, found time to examine the case and to aid and encourage us by his valuable opinion and helpful advice in the preparation of this paper. We would also thank the Officer Commanding, Royal Air Force Hospital, Aden, for providing us with facilities for the examination and investigation of this case.

REFERENCES

- ¹ GIBSON and MALET, *Jour. Anat. and Physiol.*, 1879-80, xiv, 1.
- ² TURNER, *Ibid.*, 103.
- ³ MARTIN, *Med. Record*, 1887, xxxii, 425.
- ⁴ BARILLET, *Comptes rend. Soc. de Biol.*, 1929, ci, 829; *Presse méd.*, 1929, No. 50, 817.
- ⁵ BENHAMOU, HERMANN, and LEVI-VALENSI, *Comptes rend. Soc. de Biol.*, 1929, cvii, 168.
- ⁶ SZENES, *Arch. f. klin. Chir.*, 1922, cxix, 116.
- ⁷ GREIG, *Edin. Med. Jour.*, 1926, Aug., 480.
- ⁸ MORTON and JORDAN, *Arch. of Surg.*, 1935, xxx, 647.
- ⁹ KEITH, Sir A., *Human Embryology and Morphology*, 5th ed., 1933, 472. London.

MELANOMA OF THE RECTUM*

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MELANOMA of the rectum is an uncommon disease, but probably less so than would appear from an examination of the literature. It is a condition of peculiar interest, particularly from the point of view of its pathogenesis and its relationship to the other forms of melanomata. Melanomata of the skin and the eye are well known, and rare cases arising from derivatives of the neural ectoderm, especially in the central nervous system and the suprarenal medulla, have been described. In the alimentary canal, apart from the rectum, melanomata are exceedingly rare. In his admirable and most exhaustive monograph on the melanomata, Dawson makes no specific mention of the occurrence of this tumour in the rectum, and we have not been able to find any detailed account of the disease in English literature, although a certain number of cases have been briefly recorded. We therefore feel that a recent case which we were able to study in detail should be put on record.

HISTORY

Melanomata of the rectum were recognized in the lower animals for many years before they were known in man. They were described in the horse before the end of the eighteenth century. Laennec, in 1806, isolated and described the ordinary forms of melanomata in man, but it was not until 1857 that a case of melanoma of the rectum was recorded by Moore. Virchow, in 1869, mentions a case reported by Maier, and adds that he had seen one case himself which ran a very malignant course. The first complete record of a case was that of Paneth in 1883; he described fully a personal case and mentioned 8 others recorded up to that date. From that time onwards records became more numerous, and in 1913 Chalié and Bonnet, in a very excellent and comprehensive review of the whole subject, were able to collect and analyse the records of 64 cases, including one of their own; they mention 8 other possible cases of which they had heard but which they had been unable to trace. This paper by Chalié and Bonnet is much the most satisfactory, and indeed is the only complete, article on the subject in modern literature. In 1918 Churchman in reporting a case had been able to collect only 2 more since 1913, bringing the number of authenticated cases up to 67. Since 1918 we have been able to find only 11 cases in the literature, those of Kraker, McGuire and Leahy, Goldblatt, Allan, Kallet and Saltzstein (3 cases), Landman, Gerritzen, Marino, and Ingle. This brings the total of recorded cases up to 78, with 8 others mentioned but untraced by Chalié and Bonnet.

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CASE REPORT

The following case was operated on by one of us as a malignant tumour of the rectum, but its true nature was not recognized until the routine examination of the operation specimen was carried out. The patient was a married woman, aged 53. She had complained of pain in the rectum and passage of blood on defæcation for a period of four months. Constipation was not a prominent feature, and her general health had remained satisfactory. On digital examination of the rectum an indurated swelling was encountered; it was situated at the level of the ano-rectal junction and was mainly on the posterior wall. It projected into the lumen of the bowel but did not prevent the passage of the finger. There was no enlargement of the inguinal glands, and abdominal examination did not reveal any abnormality. The condition was regarded as an adenocarcinoma of the rectum of the ordinary type.

OPERATION.—At the end of August, 1935, an inguinal colostomy was performed. The liver was examined and found to be smooth on the surface and free from any palpable evidence of secondary growths. There was no evidence of metastases in the peritoneum and no enlarged glands were found in the pelvic mesocolon or in relation to the rectum. The tumour was not adherent to the perirectal tissues and was considered to be eminently suitable for a radical operation.

On Sept. 8, 1935, under general anæsthesia, an excision of the rectum was carried out by the perineal route. The coccyx was excised, the peritoneal cavity opened freely, and the bowel divided through the lower part of the pelvic colon. The peritoneum and the proximal end of the colon having been closed, the cavity was packed with gauze and allowed to heal by granulation. The patient made a good recovery from the operation. She was examined in April, 1936, and was found to be in good health. No gross evidence of metastasis could be found.

MORBID ANATOMY AND HISTOPATHOLOGY

The true nature of the tumour was only recognized in the course of the routine examination of the operation specimen. The method employed has been previously reported. The site and size of the growth and the condition of the lymphatic glands as regards infection by the growth are recorded in the chart shown in *Fig. 47*. After dissection of the lymphatic glands a section was made through the specimen in the sagittal plane. This showed a large blackish-brown tumour arising from the upper part of the anal canal and the lower part of the rectum proper. The appearance of the cut surface of the tumour with its anatomical relationships is well shown in *Fig. 48*.

The tumour involved the posterior and lateral aspects of the bowel wall, extending over about two-thirds of its circumference. It showed a very pronounced tendency to bulge into the lumen of the bowel; in the vertical plane the attachment to the bowel wall measured 2.4 cm., while the greatest vertical measurement of the tumour was 4.7 cm. Its base appeared to rest on the muscularis of the lower part of the rectum and the upper part of the anal canal. The tumour was firm and elastic in consistence, but here and there small areas of softening were present. The cut surface of the growth presented an appearance of lobulation, and at one point on the free surface there was an umbilicated depression suggesting the appearance sometimes seen in malignant nodules on the surface of the liver. The free surface of the tumour did not show gross ulceration.

Lying posterior to the main tumour, but separated from it by the muscular wall of the rectum, there was a large rounded nodule of a brownish-black colour. It appeared to be entirely extra-rectal and we were unable to trace any direct continuity between this nodule and the primary tumour. It was enclosed in a well-marked capsule posteriorly, probably derived from the fascial covering or sheath

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of the rectum. It measured 4.1 cm. in the vertical plane and 3.4 cm. in the antero-posterior plane. Another much smaller nodule was present about the level of the middle of the base of the main tumour. It also lay outside the muscular coat of the bowel, and measured about 0.5 cm. in the vertical plane. Apart from these two nodules there was no other evidence of spread of the tumour outside the wall of the rectum. There was no pigmentation of the peri-anal skin or of the mucosa of the rectum, apart from some slight brownish discoloration in the immediate neighbourhood of the tumour.

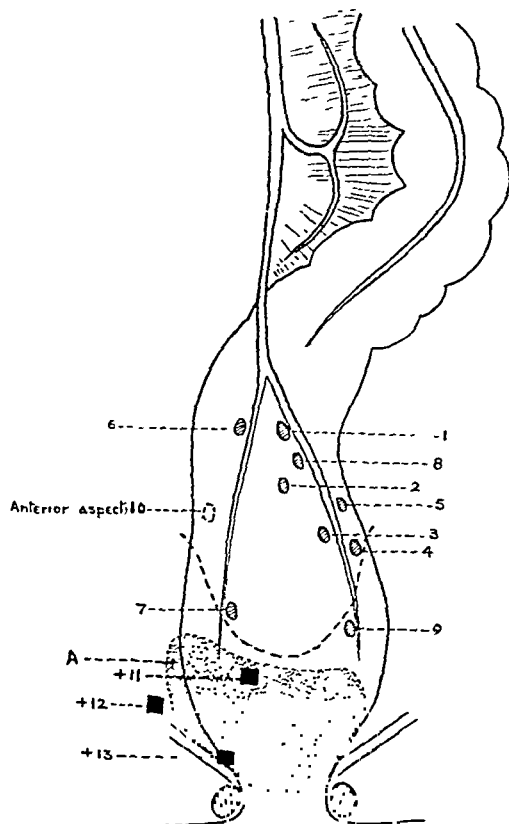


FIG. 47.—Chart showing the position of the tumour and the associated lymphatic glands.

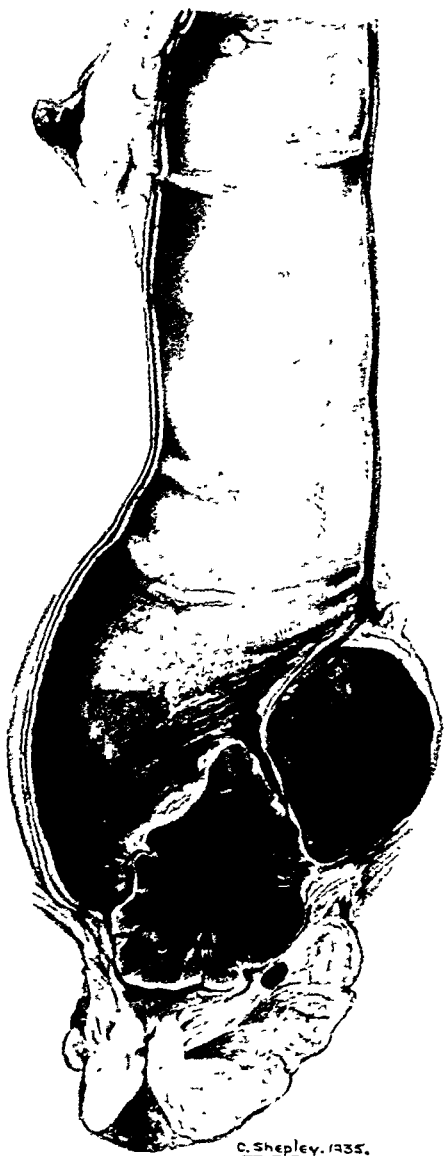


FIG. 48.—Drawing of a sagittal section through the operation specimen. The tumour is growing from the posterior aspect of the bowel.

On dissection of the peri-rectal fat, ten lymphatic glands were found, as indicated in the chart. Nine of these lay on the posterior aspect of the rectum in relation to the branches of the superior hæmorrhoidal artery; the remaining

gland lay on the anterior aspect of the rectum. All the glands were of moderate size, the largest being about 0.5 cm. in diameter, while the majority were about half this size. On external examination, only two of the glands appeared blackish

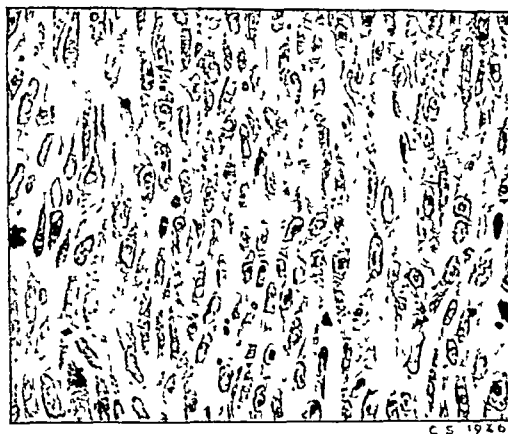


FIG. 49.—Microscopical section of the primary tumour. It is made up largely of spindle cells, presenting a resemblance to a sarcoma

in colour, but on section all the glands showed some evidence of blackish pigmentation with the exception of the one found on the anterior aspect of the rectum.

Microscopical Appearances.—There was a remarkable difference between the microscopical appearance of the primary tumour and that of the extra-rectal

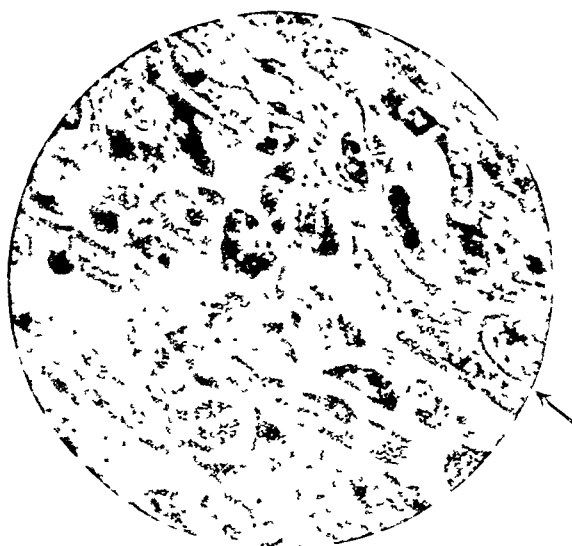


FIG. 50 —Photomicrograph of the tumour showing a large pigment-carrying cell on the right ($\times 900$)

nodule. The greater part of the primary tumour was made up of long strands of spindle-shaped cells and presented a close resemblance to the picture of a spindle-celled sarcoma (*Fig. 49*). These elongated cells contained a varying amount of

pigment in the form of fine brownish granules scattered throughout the cytoplasm. Some of the pigment was extra-cellular, suggesting that it had been liberated into the extra-cellular spaces by the breaking down of pigment-containing cells. A considerable proportion of the cells were entirely free from pigment. Although

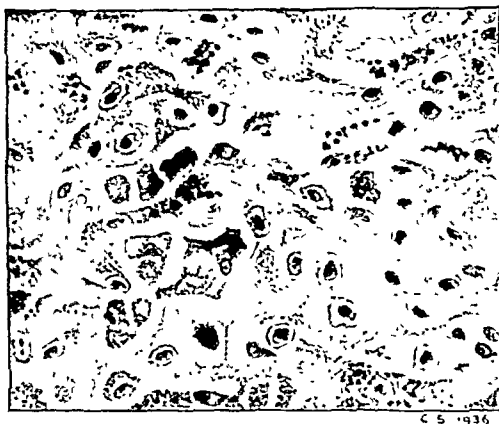


FIG. 51.—Section through the extra-rectal nodule. The cells are polyhedral in shape with abundant cytoplasm. Pigment granules are numerous.

most of the cells were arranged in strands or columns, in a few areas attempts at the formation of alveoli were seen. A second type of cell was present in small numbers, easily distinguishable from the spindle-shaped tumour cells. It was much larger than the tumour cells, being sometimes two or three times the size, and its cytoplasm was frequently packed with coarse pigment granules (Fig. 50). It has

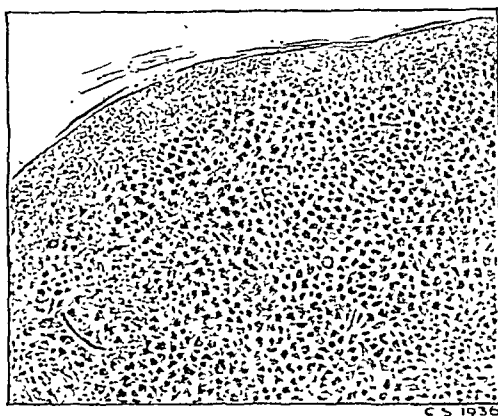


FIG. 52.—Section through one of the lymphatic glands. The lymphoid tissue has been largely replaced by pigment-carrying melanophores.

been shown that this is not a malignant cell but is a phagocytic pigment-carrier. It has been named a *chromatophore* or *melanophore* in contrast to the *melanoblast*, the pigment-producing cell. It was present also in the extra-rectal nodule and in large numbers in the lymphatic glands.

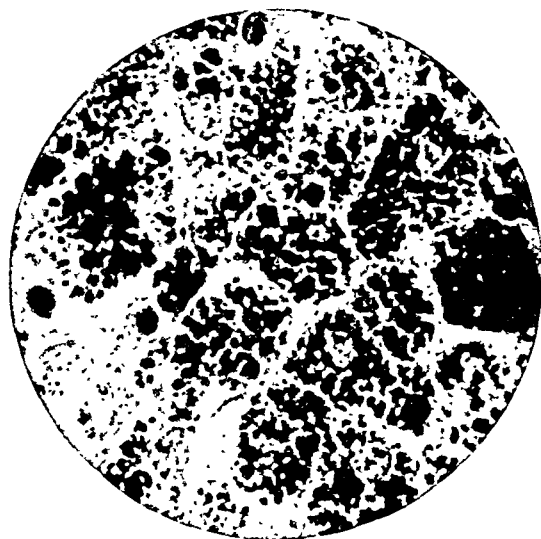


FIG. 53.—A group of melanophores from a lymphatic gland. The cytoplasm is so packed with coarse pigment granules that the nucleus in some of the cells is almost hidden. ($\times 900$.)

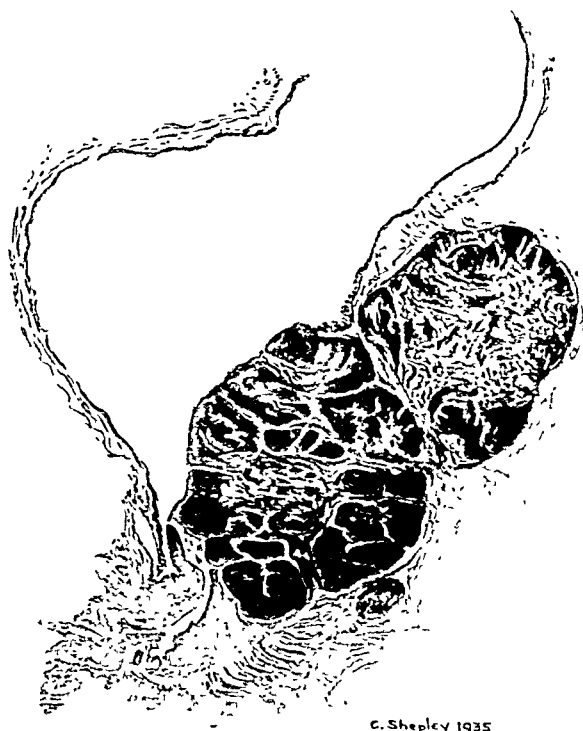


FIG. 54.—Large microscopical section through the tumour. This shows the relation of the primary tumour to the rectal wall and the general structure of the primary tumour and the extra-rectal nodule.

A fairly well-developed stroma of fibrous tissue was present, giving to the cut section of the tumour a lobulated appearance. The free surface of the growth was covered with a fibrinous exudate except at the upper and lower extremities, where the rectal mucosa and the squamous epithelium of the anal canal were intact for a short distance.

The extra-rectal nodule was made up almost entirely of an epithelial-like type of cell. The stroma was very scanty and the nodule was much more homogeneous in structure than the primary tumour. The cells were polyhedral in shape with abundant cytoplasm (*Fig. 51*). The nucleus was large and vesicular. Pigment was more abundant than in the primary tumour and was both extra-cellular and intra-cellular in position. The particles of pigment varied in size from fine powdery granules to large coarse deposits. The contrast between this epithelial

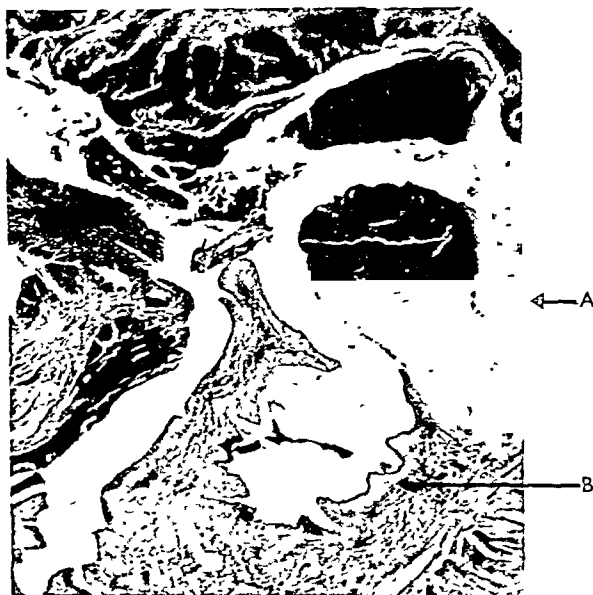


FIG. 55.—Low-power photomicrograph at the lower end of the tumour. On the right it can be seen that the tumour tissue (A) is in direct contact with the squamous epithelium of the anal canal (B). ($\times 5$.)

type of cell and the sarcoma-like cell of the primary tumour is very striking, and this polymorphism of the cells of the melanomata is partly responsible for the difference of opinion as to their histogenesis.

The lymphatic glands showed pigmentation to the naked eye in nine out of ten cases, as already explained. On microscopical examination the pigmented glands all showed replacement of the lymphoid tissue to a greater or lesser degree by large cells, many of which were loaded with pigment granules. It is not an uncommon finding to have such glands described as malignant, to be the seat, in fact, of metastatic tumour cell deposit. Harding and Passey, working with a melanoma of the mouse, which they were able to transplant with success, were never able to obtain successful grafts from the pigmented glands which accompanied these tumours. They conclude, on this and other grounds, that the large pigment-bearing

cells in the glands were not truly tumour cells. We also have concluded that the cells containing pigment in the glands were not indicative of metastatic deposit but of transference or ingestion of pigment by phagocytic cells. The importance of this view with regard to prognosis in such a case is obvious.

In one gland, No. 4 on the chart, the lymphoid tissue had been entirely replaced by these pigment-carrying cells except for a narrow strip at the surface of the gland (*Fig. 52*). In the others, with one exception, there was a varying degree of replacement. The exception was No. 10, which lay on the anterior aspect of the rectum, away from the main stream of the lymphatic return. Where only a few melanophores were present, these were mostly found at the peripheral part of the gland, as if they had just entered from the afferent lymphatics. The assumption is that the pigment which is liberated by the breaking down of malignant cells in the primary tumour is ingested by the phagocytic pigment carriers and conveyed to the regional lymphatic glands. Some of the melanophores were so packed with pigment granules that the nucleus was almost invisible (*Fig. 53*).

A number of large microscopical sections of the tumour and the extra-rectal nodule were cut in the sagittal plane (*Fig. 54*). A close examination of these does not justify a dogmatic statement as to the origin of the tumour, but it was quite evident that the tumour tissue came into extremely close relationship with the squamous epithelium of the anal canal, as shown in *Fig. 55*. The tumour can be seen to lie immediately underneath the modified skin which lines the lower part of the canal. This fact is of importance in relation to the histogenesis of the tumour.

DISCUSSION

This case may be regarded as a typical illustration of melanoma of the rectum. Clinically, the symptoms are similar to those of adenocarcinoma of the rectum, and the condition will usually be mistaken for that lesion. Blackish discharge on the examining finger, however, might arouse suspicion as to the nature of the tumour. If the speculum is introduced, the dark colour of the growth will suggest its nature. In some cases there has been pigmentation of the anal orifice, and this appearance has sometimes been confused with thrombotic external hæmorrhoids. The tumour in the present case was sessile, but in many of the recorded cases it has been pedunculated—33 per cent (Loques)—and may even present at the anus as a polypus. A melanoma bulges into the lumen of the bowel; it shows no tendency to spread in an annular fashion, causing stenosis, as is frequently the case in adenocarcinoma. If it obstructs the bowel, as it may do in the long run, it does so by a process of occlusion and not of stenosis. It resembles in this respect a sarcoma of the rectum.

Origin of the Tumour.—Much discussion has taken place as to the essential nature of the melanomata, and the custom of naming these tumours according to the form and configuration of their cells—as in the use of the terms ‘melano-sarcoma’, ‘-carcinoma’, ‘-endothelioma’, ‘-perithelioma’, and ‘-fibro-sarcoma’—was formerly, and to a certain extent still is, a cause of confusion. In one part of our specimen the appearances were those of ‘melano-sarcoma’, while in another they were those of ‘melano-carcinoma’; it is evident, therefore, that such terms cannot be applied with real consistency to this type of tumour. Most English

writers follow the recommendation of Ewing and employ the term *melanoma*, to emphasize the specific character of the tumour and its cells of origin.

Ewing summarizes the various theories as to the origin of the melanomata. It is to be noted that he uses the term *chromatophore* in the sense that we, following the recommendation of Dawson, have used the term *melanoblast*, the pigment-producing tumour cell. He states that they arise either: (1) Exclusively from mesoblastic chromatophores (melanoblasts); (2) Exclusively from epithelial cells and epithelial chromatophores; (3) From naevus cells in the skin and mesoblastic chromatophores in choroid and meninges; (4) From endothelial cells of blood- and lymph-vessels, or of nerve-trunks; (5) From chromatophores, tactile corpuscles, and nerve-cells forming the end-apparatus of the cutaneous sensory nerves (Masson). Masson has brought forward very strong evidence in support of his view. Dawson, in his paper on the melanomata, expresses his belief that the basal cells of the epidermis are the true pigment producers; he traces the origin of quiescent naevus cells from the basal epithelium by a process of emigration and depigmentation; he derives all the histological varieties of cutaneous melanomata from the original basal cells; uveal melanomata, he states, are embryologically derived from the pigmented retinal epithelium; and melanomata of adrenal and other deep organs from pigmented neuro-epithelium, postulating a possible dispersion of ectodermal pigmented cells along the paths of out-growing nerves.

We do not feel competent to discuss a subject presenting such very great histological difficulties, but we should like to emphasize the fact that melanoma of the rectum almost always occurs low down, either in contact with the epidermal portion of the anal canal or close to it. Chalié and Bonnet found that in exceptional cases it might be as much as 5 to 6 cm. above the anus, i.e., 1 to 2.5 cm. above the anal canal, but even at this level the tumour may have been related to displaced portions of epidermis. The almost uniformly low position and the exceeding rarity of melanomata in other parts of the alimentary canal suggest that it probably arises from the skin of the anal canal. Chalié and Bonnet, after a most exact histological examination of their own case and a careful analysis of the 63 cases which they had collected, came to the conclusion that "primary melanotic tumours of the ano-rectal region are cutaneous epitheliomata from the anal canal, capable clinically of becoming tumours of the rectum by progressive infiltration along the length of the submucosa upwards". These writers believed that they were able to trace the exact point of departure of the tumour from the Malpighian epithelium of the anal canal. We were not able to do this in our specimen, but we observed that the tumour tissue came into the closest relationship to the squamous epithelium of the lower part of the anal canal. The evidence afforded by a study of melanoma of the rectum seems to favour the origin of this tumour from skin; we refrain from discussing whether it is more likely to arise from the basal cells of the epithelium (Dawson) or from the end apparatus of sensory nerves (Masson).

Spread of the Tumour.—Melanoma of the ano-rectal region invades the submucosa and shows a pronounced tendency to grow upwards into the rectum, which is presumably the direction of least resistance. It shows no special tendency to spread in a circular manner, but commonly bulges into the lumen of the bowel. At the same time it spreads deeply, and in our case has spread through the muscularis and invaded the perirectal fat. The layer of fat is enclosed in the fascial sheath of

the rectum, and this fibrous membrane, as happens in adenocarcinoma of the rectum, appeared to have contained or limited the spread of the growth. In the rectum, as elsewhere, melanotic tumours may be exceedingly malignant and may become generalized at an early stage, spread taking place both by blood-stream and by lymphatics. Secondaries have been found in almost every organ in the body, the liver being the most common site. Enlargement of the inguinal glands may be an early indication of metastasis. In our case there were one or two points which seemed to suggest a comparatively low degree of malignancy. There was no evidence of spread to lymphatic glands and no recognizable sign of spread to the liver. Although the tumour had spread through the muscularis, the extra-rectal growth presented appearances which might be interpreted as indicative of a lesser degree of malignancy than the primary growth. Harding and Passey have shown that the more malignant the melanoma the less pigment does it contain. The extra-rectal nodule showed more abundant pigment than the primary growth, and, its cells being large and epithelial in type, it might be considered to show a lesser degree of metaplasia. Sir David Wilkie has kindly supplied us with the notes of a case which are of interest in this respect. The patient, an elderly female, was operated on on April 29, 1933, and a papillomatous tumour of the rectum was removed by local excision; this proved on subsequent microscopical examination to be malignant. Two nodules were present in the neighbourhood, one of which was outside the wall of the bowel. It was felt that a radical operation was unlikely to be successful, and radium needles were inserted on May 15, 1933, after removal of the coccyx. The patient returned in June, 1934, with a recurrence of her symptoms. A small tumour was present in the wall of the rectum, which was found on microscopical examination to be a melanoma. The patient was again treated by radium. She returned in March, 1935, with extensive local recurrence in the rectal wall and perirectal tissues but without any evidence of general dissemination. A large dose of radium was again inserted extra-rectally. A bad prognosis was given, but in January, 1936, the patient reported in good health with no rectal trouble and having gained 18 lb. in weight. This case shows that dissemination of the tumour may be delayed for a long time, and also that these tumours may be very radio-sensitive, which lends support to the theory of baso-squamous epithelial origin. It is evident, therefore, that, where a radical operation is feasible, it must in some cases afford a reasonable prospect of success. However, until a large number of cases of melanoma of the rectum have been recorded and followed up it will not be possible to form an accurate estimate of the degree of malignancy of this type of tumour.

We have already referred to the misleading appearance of the lymphatic glands in this case. The pigmentation on naked-eye examination and the infiltration microscopically with large phagocytic pigment-carrying cells gave a false impression of malignant invasion.

CONCLUSIONS

1. The melanoma of the rectum is derived from the stratified squamous epithelial portion of the anal canal.
2. Regional lymph nodes which contain pigmented cells are not necessarily the seat of metastasis.

We desire to express our thanks to Sir David Wilkie for his advice and encouragement and for the facilities which he has given us in the Department of Surgical Research Laboratory. We are also much indebted to Colonel W. F. Harvey for his kindly interest in this paper. We wish to thank Mr. C. Shepley for the great care and skill with which he has executed the coloured illustrations.

BIBLIOGRAPHY

- DAWSON, *Edin. Med. Jour.*, 1925, xxxii, 509.
CHALIER and BONNET, *Rev. de Chir.*, 1912, xlv, 914.
CHURCHMAN, *Amer. Jour. Med. Sci.*, 1918, clv, 639.
KRAKER, *Trans. Amer. Proc. Soc.*, 1925, 102.
MCGUIRE and LEAHY, *Bull. Buffalo Gen. Hosp.*, 1924, ii, 85.
GOLDBLATT, *Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 1986.
ALLAN, *Trans. Amer. Proc. Soc.*, 1931, xxxii, 31.
KALLET and SALTZSTEIN, *Arch. of Surg.*, 1933, xxvi, 633.
LANDMAN, *Trans. Amer. Proc. Soc.*, 1933, xxxiv, 65.
GERRITZEN, *Arch. f. klin. Chir.*, 1933, clxxviii, 400.
MARINO, *Jour. Amer. Med. Assoc.*, 1934, cii, 203.
INGLE, *Indian Med. Gaz.*, 1935, lxx, 266.
WOOD and WILKIE, *Edin. Med. Jour.*, 1933, xl, 321.
HARDING and PASSEY, *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 417.
EWING, *Neoplastic Diseases*, 3rd. ed., 1928.
MASSON, *Ann. d'Anat. pathol.*, 1926, iii, 417.

RADICAL EXCISION OF MALIGNANT TUMOURS IN THE ETHMOID-MAXILLARY REGION

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AN opportunity occurred recently of examining a patient who was treated twelve years ago by a radical operation for an extensive malignant tumour in the left maxillary and ethmoidal regions. It is the purpose of the present paper to describe the operation employed in this case and to show that such an extensive procedure may have a limited but real field of usefulness.

CASE REPORT

The patient was an agricultural labourer, aged 44, who was admitted to hospital on Aug. 17, 1923, complaining that the left side of his nose had been blocked for the previous three months. Shortly after he noticed this, the left side of his face began to swell. During the first six weeks he had experienced pain at the back of the left eye, but this had subsequently disappeared.



FIG. 56—A, Appearance of the patient at the present time, B, The eye-shade removed. The sphenoidal sinus is visible at the back of the orbit.

On admission his general condition was good. There was a marked swelling of the left side of the face. The skin of the cheek was red, œdematous, and fixed to the maxilla, and on palpation areas of fluctuation were found. The eyeball was considerably proptosed, but the palpebral fissure was occluded by a marked œdema of the eyelids, especially of the lower. The left nostril was completely blocked by a mass arising from the lateral wall. The left half of the hard palate was depressed into the mouth, forming a rounded, fluctuating swelling, and there was a hard, smooth mass to the outer side of the left upper alveolus. No enlarged lymphatic glands were felt in the neck.

An X-ray film showed irregular opacities in the left nasal fossa, left antrum, left frontal and left ethmoidal sinuses suggestive of malignant disease.

A biopsy from the mass in the nostril was performed and a section showed that the growth was an endothelioma.

On Oct. 17, 1923, a radical operation for the removal of the growth was performed by Mr. Wilfred Trotter. After a preliminary laryngotomy the anæsthetic was given through the laryngotomy tube and the pharynx carefully plugged. The whole tumour, together with a margin of normal tissue, was removed by the method described in detail below. During the operation the frontal and ethmoidal sinuses were opened and found to contain polypoid material. Recovery was uneventful and the patient left hospital six weeks later.

When seen on Dec. 12, 1935, over twelve years later, the patient was in excellent general health and spirits. There was no sign of local recurrence or metastasis. He wore an eye-shade to cover the defect in the base of the orbit, but otherwise the external deformity was minimal (*Fig. 56*). He stated that his acquaintances believed that the eye-shade concealed a 'weak' eye.

On inspection of the buccal cavity an oval recess 3 cm. by 2 cm. was visible at the site of the left upper alveolus and left half of the hard palate. The soft palate was intact, but somewhat drawn up on the left side. Through the gap in the facial scar the nasal septum was visible medially, and behind this the left sphenoidal sinus. Inferiorly the left half of the soft palate was visible with the nasopharynx above and behind and the tongue below and in front.

The patient had been perfectly well and free from pain since recovery from the immediate effects of the operation. A plastic operation for the closure of the naso-orbital defect has been suggested to him; but as he would still have to wear an eye-shade he is disinclined to bother about it.

THE OPERATION

The object of the operation is to remove intact the whole of the outer wall of the nose (*Figs. 57, 58*). The most inaccessible part of this mass of tissue is the lateral mass of the ethmoid, where experience shows that many of the growths of this region originate. The lateral mass of the ethmoid cannot be removed intact unless it is possible to sacrifice the contents of the orbit. In the advanced cases for which the operation is particularly designed, it is already clear from the proptosis and possibly chemosis that the normal position and function of the eye cannot be preserved or regained, and there is no objection to the approach through the orbit, which is an essential step in the extirpation of the ethmoid. Many of the growths originating in the lateral mass of the ethmoid involve the maxillary antrum comparatively late, and in these cases the hard palate may be preserved without risk.

The operation is definitely designed for advanced cases, and the surgeon should beware of concluding too readily that a given case is inoperable. Much of the apparently hopeless aspect of the case reported here was due to the severe secondary inflammation of the accessory sinuses. Spread of the disease to the tissues of the apex of the orbit and the foramina there is perhaps the commonest cause of failure in apparently hopeful cases.

The incision is of the Ferguson type, but enters the palpebral fissure at the inner canthus and leaves it at the outer canthus to be prolonged along the zygoma.

The whole of the soft tissues in the infero-lateral flap, if not involved in the growth, is raised from the bone and thrown outwards. In cases where involvement of the antrum is doubtful a small opening is made in the anterior surface of the maxilla and the condition of the cavity decided by inspection. The upper flap, including

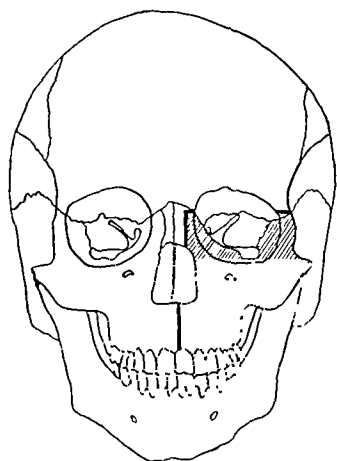


FIG. 57.—Outline of skull showing sites at which bone is divided. The portion to be excised is shaded.

the upper eyelid, is next dissected up, the external angular process of the frontal divided, and the zygoma cut through, or, better, the middle portion removed. The outer wall of the orbit is now exposed and removed, giving free access to the orbital contents. The latter are carefully separated by working back to the apex of the orbit until the extreme posterior end of the inner wall is exposed. They are also cleared from the roof, remaining attached only to the lateral mass of the ethmoid and the maxilla with which they will be removed.

If the antrum is invaded by the growth, the next step is to divide the alveolus and the hard palate close to the septum, subsequently separating off the soft palate. When the antrum is free the corresponding cut is made horizontally across the maxilla above the level of the hard palate, which is preserved.

After a saw cut has separated the nasal bone from the nasal process of the maxilla, the separation of the lateral mass of the ethmoid from the frontal can be undertaken. A chisel driven directly backwards at the level of the orbital roof effects this division, the frontal sinus being opened in the process. The mass which has thus been outlined is now gently levered loose from its remaining bony attachments posteriorly.

The excised parts, which should come away in a compact unbroken mass, include the maxilla, the lateral mass of the ethmoid, the malar bone, and the orbital contents, and on inspection of the inner surface the whole of the lateral wall of the nasal fossa is visible.

DISCUSSION

The present paper is intended to deal with only one limited part of the large topic of naso-maxillary malignant disease. The case described illustrates, however, certain of the more general aspects of the subject, which may therefore be briefly referred to.

Secondary suppuration and polypoid disease of the accessory sinuses is a well-known complication of malignant disease in the region under discussion. It was very severe in the case described, causing profuse purulent discharge and much brawny swelling of the cheek. No doubt it is due to obstruction of the sinuses

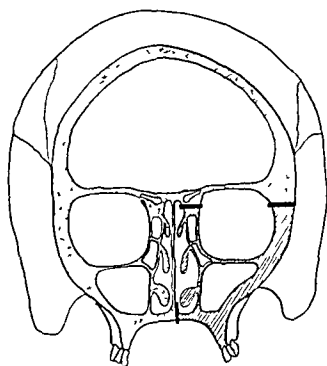


FIG. 58.—Coronal section of skull, diagrammatic, to show the relations of the divisions to the surrounding structures.

by the growth. It is an important complication in two respects. First, it may so much aggravate the severity of the picture produced by the tumour itself as to make an operable case seem utterly hopeless. Secondly, it adds greatly to the difficulties of applying radiotherapy to these cases. There seems, moreover, little doubt that the presence of secondary inflammation favours rather than discourages the spread of the primary neoplasm.

The site of origin of the tumour is of the first importance in designing the treatment of any given case. In former times it was apt to be assumed much too readily that growths of the class under consideration have their origin in the maxilla. The result was that they were treated by a formal excision of the upper jaw without the least prospect, in many cases, of any good being done. More exact methods have shown that while in many cases the growth does begin in the maxillary antrum, or penetrate to it from the alveolus, probably an equal number begins in the outer wall of the nose in relation to the lateral mass of the ethmoid. It is for dealing with these latter cases that the operation described above is suitable. The precise diagnosis of the site of origin of the tumour is therefore very important, though not always attainable. In the present case the early blockage of the nostril, and the pain behind the eye, were regarded as pointing to a growth arising in the lateral mass of the ethmoid.

It may be interesting to quote the opinion of three recent authors on the problem of treating maxillary-ethmoidal malignant growths. Quick¹ (1930) states that it is doubtful if a radical operation conforming to general surgical principles for the treatment of malignant disease is possible in this region. Davis² (1934) concludes that the difficulty of early diagnosis and the inaccessibility of ethmoidal carcinoma makes it doubtful whether such a mutilating operation is worth while. Harmer³ (1935), in an able presentation of the case for combined partial excision and radiotherapy, reaches the conclusion that approach should never be made through the skin. It may be agreed that in highly malignant radio-sensitive growths complete local regression is obtainable by the combined method, but is too commonly followed by death from distant metastases. In radio-resistant types the course following any type of irradiation is too frequently persistence of the growth accompanied by severe and intractable pain.

It seems clear that in cases conforming to the indications laid down the method of radical extirpation here described has certain advantages over other procedures. Suitable cases must necessarily be few, and their most important characteristics will be that the disease is in a fairly advanced stage and that it shows evidence of having originated in the outer wall of the nose.

SUMMARY

1. A case of successful radical excision of an extensive malignant tumour in the maxillary and ethmoidal regions is described.
2. The operative procedure is described.
3. An attempt is made to define the conditions in which such an operation may be the best treatment.

REFERENCES

- ¹ QUICK, D., *Radiology*, 1930, xiv, 191.
- ² DAVIS, E. D. D., *Brit. Med. Jour.*, 1934, i, 53.
- ³ HARMER, W. D., *Lancet*, 1935, i, 129.

SPONDYLOLISTHESIS

BY JAMES A. JENKINS, NEW ZEALAND

IN recent numbers of the BRITISH JOURNAL OF SURGERY three papers have appeared on this interesting condition. Shore¹ describes this deformity as found in the Bantu races of South Africa, and the developmental anomalies which predispose to the condition are discussed. Brailsford² gives a comprehensive review of the subject and reports several cases. He concludes with the following statement:—"A study of the specimens and radiographs in my cases negatives the suggested possibility of manipulative reduction of the displacement." Capener³ similarly reviews the literature and bases his paper on the study of three cases. In discussing the surgical treatment he points out that while bone-grafts undoubtedly assist many patients, they do not, from the mechanical point of view, possess great efficiency. "Tremendous forces are brought to bear upon the graft, which is badly placed for the prevention of slipping." "*The ideal operation would be an anterior bone-graft so placed as to fix the body of the fifth lumbar vertebra to the sacrum and forming a buttress or some form of antero-posterior fixation of the two halves of the divided vertebra.* The technical difficulties of such a procedure, however, preclude their trial."*

In view of the fact that nothing can be added to the developmental, clinical, and radiological aspects discussed in the papers that have recently appeared, and as complete bibliographies are appended thereto, repetition would be wearisome. No paper has indicated a course of treatment, either conservative or surgical, that can arouse any enthusiasm for end-results.

In the case here reported a gross deformity was reduced, and the method of fixation followed was that suggested by Capener as the ideal operation though impracticable on account of technical difficulties.

The deformity in an advanced case of spondylolisthesis is marked and the disability very great. Pessimism both as regards reduction of the deformity and means of adequate fixation permeates all papers. Jackets and various supports are suggested, but it is obvious that these cannot be effective if the patient is to lead an active life. Bone-grafts on the Albee principle are mentioned and the obvious mechanical inefficiency of these is referred to. The fact that the division in the vertebra is through the arch, and the graft therefore does not directly fix the bone that is slipping, makes it apparent that only imperfect fixation of the body can be achieved by this indirect means.

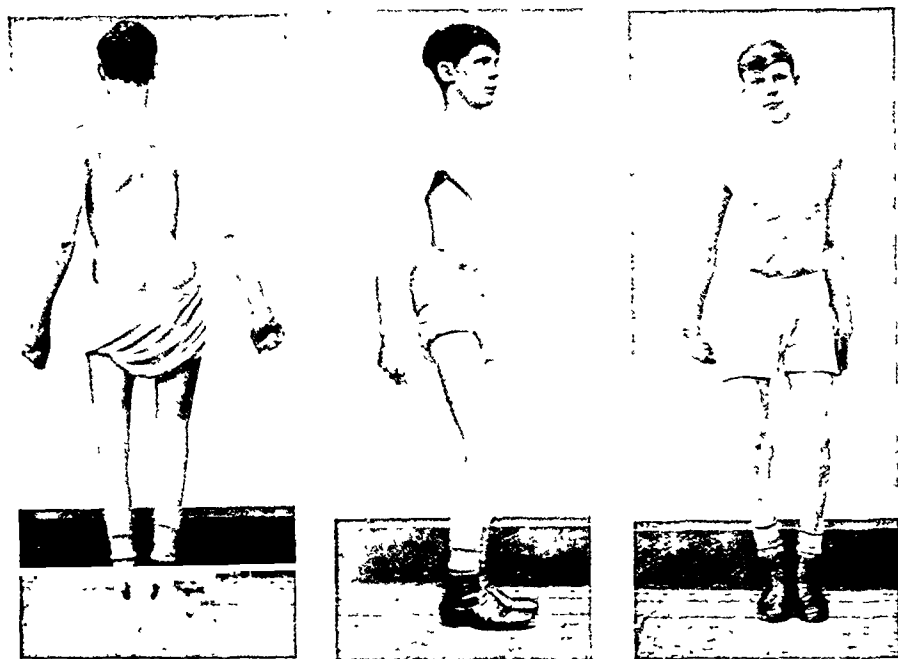
A graft consolidating the third and fourth lumbar vertebra together with the separated posterior arch of the fifth lumbar vertebra and the sacrum must do something towards stabilization of this region, but the tremendous stress when there is complete overlapping of the fifth lumbar vertebra on the first sacral is so great that the bad results reported can be readily understood.

* A case of spondylolisthesis in a boy of 14, of traumatic origin, was reported by Burns in *The Lancet*, June 10, 1933. He was treated by driving an autogenous tibial graft from the abdominal aspect of the 5th lumbar vertebra into the sacrum, with success (Ed.).

Albee states that the fourth and fifth lumbar vertebræ tend to pull themselves away from the graft in an antero-posterior direction. Brailsford quotes Turner and Tchirkin: "It is doubtful whether operative measures can aid conservative treatment in alleviating the symptoms and arresting the progress of the ailment." He also goes on to say that a study of the specimens and radiographs negatives the suggested possibility of manipulative reduction of the displacement.

CASE REPORT

A male, aged 16 years, complained of a 'bad hip'. Deformity commenced a year previously and had become progressively worse. He was easily tired, much handicapped by the posture he was forced into, and suffered pain in the lower part of the back after exercise. He could remember no previous accident, but had played the usual school games and suffered the customary injuries.



FIGS. 59-61.—Photographs showing the deformity prior to operation.

Examination showed the deformity characteristic of advanced spondylolisthesis. The posterior projection of the pelvic girdle, the forward direction of the lower lumbar spine, the shortening and forward displacement of the lumbar spine, and the compensatory lordosis, are well shown in *Figs. 59-61*.

A radiograph (*Fig. 62*) taken when the patient was first seen showed a complete displacement of the body of the fifth lumbar vertebra, its lower border lying opposite the second sacral vertebra. After admission to hospital, reduction was attempted by continuous extension. The pelvis was suspended by a sling to an overhead frame. By means of Sinclair's glue extension was applied to both legs and pelvis at the one end and to the chest at the other: 60 to 70 lb. weight were applied. The patient was encouraged to manipulate his own pelvis at frequent intervals by catching the iliac crests in his hands and forcibly pushing them away. On the fourth day he was conscious of a grating noise and a sense of change of position, and



FIG 62—X-ray of patient when first seen (Sept 29, 1934).



FIGS 63, 64—Showing the position after reduction and the way in which extension was applied.

examination showed that the deformity had largely disappeared. *Figs. 63 and 64* show the position after reduction and the way in which the extension was applied. An X-ray (*Fig. 65*) taken at this time showed that the lower surface of the fifth lumbar vertebra was now resting on the upper surface of the sacrum, but there was about half a vertebra overlap in an anterior



FIG 65.—X-ray taken on Nov. 26, 1934.

posterior direction. No further improvement was achieved by manipulation and extension, though it was carried out for a further period of two weeks.

OPERATION (Dec. 10, 1934).—The anæsthetic was rectal paraldehyde followed by novocain spinal anæsthetic supplemented by light ether anæsthesia. The abdomen was opened



FIG. 66.—X-ray taken after operation (Feb. 8, 1935).

by a right paramedian incision extending from above the umbilicus to a little above the pubes. The intestine was packed off and the sacral promontory exposed. The peritoneum over the promontory was incised for 3 in and the nerve plexus, left common iliac vein, and bifurcation

of the aorta were defined. The anterior long ligament of the spinal column was divided for $\frac{3}{4}$ in. over the anterior aspect of the fifth vertebra. Slight oozing was encountered here, and this was a little troublesome throughout the operation. With a bone-drill $\frac{1}{2}$ in. in diameter a hole was made through the body of the fifth lumbar vertebra into the anterior aspect of the first sacral vertebra for a distance of a little more than 2 in. A bone-graft cut from the tibia with an Albee saw was driven into the track made for it, a very tight and firm fit resulting. It was not found possible, as was hoped, to close the anterior longitudinal ligament over the graft. Horsley's wax was used to stop slight bone oozing. The posterior parietal peritoneum was closed and the abdomen sutured in the usual way. The operation took an hour and a quarter and the patient's condition gave no cause for anxiety at its termination. Apart from a pulse ranging from 110 to 120 for a few days convalescence was uneventful.

SUBSEQUENT PROGRESS.—Radiographs taken on Feb. 8 (*Fig. 66*) showed the graft pinning the fifth lumbar vertebra to the anterior aspect of the body of the first sacral vertebra. A plaster spinal support was applied and the patient allowed up. The support was removed three



FIGS. 67-69.—Photographs of the patient a year after operation.

months later, and since then ordinary activities have been carried on without the use of any support.

Photographs (*Figs. 67-69*) of the patient's condition a year after the operation show that the deformity has not been completely reduced but that very marked improvement has taken place. He can now walk without difficulty and pain.

Whether the surgical procedure here outlined will become the method of choice in the treatment of these cases can only be known when a large series has been done. The strain on the graft, though considerable, is not as great as would appear, as the inability of the body of the fifth lumbar vertebra to slip forward

ensures that most of the weight is still transmitted through the upper surface of the body of the first piece of the sacrum. The operation presents difficulties and risks much greater than those encountered in a posterior graft of the Albee type and results will have to be proportionately better if it is to be adopted in the treatment of this very difficult condition.

In a future case, in addition to the graft, an attempt will be made to increase the bone union between the fifth lumbar vertebra and the sacrum by elevating bone-flaps and getting direct bone union between the vertebræ themselves.

SUMMARY

1. A method of reduction of the deformity in spondylolisthesis is described.
2. Fixation by a bone-graft through the bodies of the vertebra has proved an efficient means of stabilization.

REFERENCES

- ¹ SHORE, *Brit. Jour. Surg.*, 1929, xvi, 431.
- ² BRAILSFORD, *Ibid.*, 562.
- ³ CAPENER, *Ibid.*, 1932, xix, 374.

THE CLINICAL MANIFESTATIONS OF THE SPREAD OF CARCINOMA OF THE ŒSOPHAGUS OBSERVED DURING LIFE

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THE following observations are based on a series of 100 personal cases of carcinoma of the œsophagus which have been examined and treated during the past seven years. Post-cricoid and gastro-œsophageal neoplasms have not been included, and the series is confined entirely to growths affecting the œsophagus proper.

The analysis of the case histories was instigated by the considerable literature which has accumulated within recent years on the actual, experimental, and theoretical methods of surgical removal of malignant tumours of the gullet, and the feature that became manifest from this analysis was the large number of cases which showed evidence of spread of the growth at or about the time of the first examination.

Apart from the inaccessibility of the œsophagus, its intimate relationship to other vital structures, and its poor response to surgical trauma, the possibility of radical surgical treatment of œsophageal neoplasms would appear to depend upon two debatable but related factors—namely, early diagnosis and virulence of the growth.

DIAGNOSIS

Diagnosis invariably is late, as it is dependent upon the onset of symptoms; and it is well recognized that unfortunately the early symptomatology of cancer of the œsophagus is vague, and its manifestations are apt to be regarded as trivial and unimportant. There is of necessity a latent period between the earliest carcinomatous formation and the patient's awareness of something amiss, and the duration of this period at present cannot be estimated, although serological methods may in the near future yield information on this point.

The most common complaint that causes patients to seek advice is difficulty in swallowing. Of the present series of 100 cases, 91 came complaining of dysphagia, 4 of pain, 1 of hoarseness, 2 of impaction of foreign body, 1 of glands in the neck, and 1 of hiccup. Watson¹ states that of a series of 506 patients suffering from carcinoma of the œsophagus who were admitted to the Memorial Hospital, New York, in 64 per cent dysphagia was the first symptom; and Chevalier Jackson² records that of 671 such cases, in 649 difficulty in swallowing first drew attention to the disease. The dysphagia is caused by an actual narrowing of the œsophageal lumen by tumour formation with or without a superadded muscular spasm, and so by the time this symptom appears the growth must have extended into the lumen sufficiently to interfere with the passage of food down the gullet, and it is reasonable to assume that it has spread also in other directions. As a rule the onset and

progress of the dysphagia is insidious or intermittent, and patients unconsciously masticate more thoroughly, take soft foods, and aid deglutition by copious draughts of fluids. Occasionally attention is drawn suddenly to the œsophageal lesion by the impaction of a bolus of food in the strictured area of the œsophagus; this occurred in two cases of the present series. It is surprising through what a small channel any well-masticated food may be swallowed without apparent difficulty; thus Jackson³ states that a lumen of 5 mm. affords quite an efficient food conduit.

Even when dysphagia has become well established, patients fail to obtain expert advice, and valuable time is lost. Responsibility for this loss is not due entirely to ignorance, carelessness, or fear on the part of the patient, as not infrequently they have been treated by their medical attendant for 'nervous spasm' or 'dyspepsia' for two or three months before the nature and gravity of the complaint was realized. Jackson² records that of 110 cases of œsophagoscopically proven malignant disease of the œsophagus, in 87 an inferential diagnosis of neurotic condition had previously been made.

Of the present series, the average duration of symptoms before help was sought was 3·4 months—the longest period was 11 months, and the shortest was 14 days. Abel⁴ states that the average duration of symptoms before a case comes to the surgeon is from 6 to 8 months. In Souttar's⁵ series the average time lost in this manner was 5·2 months. Broders and Vinson⁶ estimate this period as 7 months, and Watson¹ as 5·8 months. The average period between the onset of symptoms and diagnosis, therefore, is almost 6 months, and this may be even a month or two longer, as patients are notoriously inaccurate when recounting the duration of their symptoms.

VIRULENCE

In regard to the virulence of œsophageal carcinomata, opinions differ very widely, and Grey Turner,³⁷ after long observation, has stated 'that in the œsophagus as in other parts of the body, malignant disease varies in type and in behaviour.' Several observers regard the growths as relatively benign, and Abel,⁷ who reviewed the findings of numerous investigators, concluded that at least one out of every four cases is operable by radical surgical methods—even at the time of death. Other writers (Wright,⁸ Musgrave Woodman,⁹ and Chevalier Jackson²) consider the tumours to be of low virulence so long as they are confined to the œsophagus itself, but that once they transgress these boundaries their growth is rapid and destructive. Lastly, there is a growing volume of opinion that œsophageal neoplasms are very virulent lesions. Vinson,¹⁰ reviewing 1000 cases of carcinoma of the œsophagus which were seen at the Mayo Clinic during a ten-year period, concludes that the condition is very highly malignant, as indicated by its rapidly progressive clinical course. Presumptive evidence of their virulence is afforded by the results of a recent investigation by Roberts,¹¹ who analysed the post-mortem findings of a large number of patients who had died from malignant disease of various regions. He found that the gravity of the case bore a definite relationship to the proximity of the tumour to the mediastinal lymph-glands, particularly to the tracheo-bronchial group.

The rapidly killing properties of malignant œsophageal tumours may be gauged by the following observations. Watson,¹ in a series of 208 cases, found that the

average length of life after the appearance of the first symptoms was 10·5 months, and after application to hospital 4·83 months. From Souttar's⁵ published cases an analysis shows that the average survival period from the onset of the first symptoms was 10·5 months, and that from the time of application to hospital was 4·7 months. Of the present series, only 57 cases have been followed to death up to date, and of these the average duration of life from the first symptoms was 9·8 months, and that from the time of application to hospital was 5·9 months. Clayton¹² estimates the average length of life from the appearance of the first symptoms to be 7 months. Chevalier Jackson,² on the other hand, states that if patients suffering from carcinoma of the œsophagus have an abundance of water and a full allowance of a well-balanced diet, they will survive for at least 2 years—several have been known to live for 5 years, and one for 6 years.

Post-mortem examination of patients who have died from carcinoma of the œsophagus frequently reveals widespread local and distant extension; at other times no macroscopic secondaries are manifest. Souttar⁵ and Watson¹ state that the virulence of the growths is such that they frequently kill the patient before there is time for the formation of secondary tumours. This statement is borne out by the observation that in a series of 70 autopsies on patients who died from carcinoma of the œsophagus, Helsley¹³ found that those who had no demonstrable metastases had had symptoms of an average duration of 4·5 months only, which is about half the average survival time from the onset of symptoms for all cases. Ewing¹⁴ is of the opinion that such growths form early and extensive metastases, and Illingworth and Dick¹⁵ state that fully 50 per cent of such cases which come to post-mortem show spread by the lymph-vascular system. Of the present series, only 13 cases have come to autopsy, and of these 10 showed metastases. Clayton¹² states that, of 41 post-mortem examinations, in 21 he found metastases below the diaphragm, and in 11 of these the liver was involved. Souttar⁵ reports macroscopic secondary growths in 8 cases out of 18; Watson¹ in 14 out of 27; and Broders and Vinson⁶ in 27 out of 42 post-mortem examinations.

Histologically carcinomata of the œsophagus are mostly of a highly malignant type when graded by Broders' method. *Table I* shows the findings of three groups of observers:—

Table I.—RELATIVE FREQUENCY OF DIFFERENT GRADES OF ŒSOPHAGEAL CARCINOMA

OBSERVER	NO. OF CASES	GRADE I	GRADE II	GRADE III	GRADE IV
Broders and Vinson ⁶	207	—	16	95	96
Watson ¹ ..	202	15	148	39	—
Clayton ¹² ..	39	3	15	12	9
Percentages	—	4%	40%	32·5%	23·5%

It is of interest to note also that Cronin Lowe¹⁶ has found by his modification of the Bendien test that patients with carcinoma of the œsophagus show a more

markedly positive serological reaction than do patients developing carcinoma elsewhere ; and he has kindly allowed me to quote his unpublished data :—

- 12 *Carcinoma of Breast*.—Early cases, clinically and surgically diagnosed as such. Average reaction, 27·5.
- 12 *Carcinoma of Cervix Uteri*.—Early cases. Average reaction, 27·5.
- 12 *Carcinoma of Rectum and Colon*.—Early cases, many of them removable by excision. Average reaction, 27.
- 12 *Carcinoma of Stomach*.—Early cases, several treated by gastrectomy. Average reaction, 26.
- 12 *Carcinoma of Œsophagus*.—Average reaction, 24.

From these observations he suggests that carcinoma of the Œsophagus may insidiously develop to a greater degree of malignancy before clinical diagnosis is obvious than is the case in the development of carcinoma in the other areas referred to in the table ; his findings also emphasize the fact of late diagnosis.

MODES OF SPREAD

Of the 100 cases under review, 29 showed definite clinical evidence of extension in or beyond the Œsophagus at or about the time of the first examination. The possible modes of spread of Œsophageal carcinoma are by one or more of the following routes : (1) Direct extension ; (2) Lymphatic permeation and embolism ; (3) Blood-stream ; (4) Implantation. Abel⁷ describes a further mode—by physiological propulsion—but this can be included under the heading of implantation for the purpose of these notes.

DIRECT EXTENSION

Symptoms and signs of spread by direct extension will naturally depend upon the level of the growth. In its upper third the Œsophagus is in close contact with the trachea, recurrent laryngeal nerves, carotid arteries, and pleuræ. In its middle third with the trachea, left bronchus, intercostal arteries, thoracic duct, hemi-azygos veins, aortic arch, left subclavian artery, and right pleura. In its lower third with the left auricle, pericardium, descending thoracic aorta, and pleuræ. Thus the Œsophagus in its upper two-thirds is in relation to many important structures (*Fig. 70*), and spread from growths situated in this locality is more likely to give rise to signs and symptoms than from those situated below the tracheal bifurcation.

Excluding post-cricoid and gastro-Œsophageal growths, the most frequent site of Œsophageal carcinoma is still undetermined. *Table II* gives the percentage location recorded by several observers, and it would appear from this analysis that a large proportion of growths occur in the middle third of the Œsophagus.

Recurrent Laryngeal Nerve.—The most common manifest complication of spread in this series was caused by involvement of the recurrent laryngeal nerves. This finding is in accord with the observations of numerous writers, and it is of interest to note that such cases supplied the data on which Semon²¹ based his law fifty years ago. Pressure on or destruction of the recurrent laryngeal nerve by actual growth or enlarged glands is characterized by hoarseness, aphonia, cough, dyspnœa, and paralysis of the vocal cord or cords when viewed by the laryngeal mirror. Hoarseness due to vocal-cord paralysis must be differentiated from the

hoarseness of a laryngitis caused by overflow of mucus, etc., into the larynx. This latter complication has been stressed by Chevalier Jackson,³ and is mentioned later.

There were 13 patients of the present series who exhibited vocal-cord paralysis when they first applied for treatment. In 8 cases the left cord was affected, in 4 the right cord, and in 1 case both cords were immobile. Cases due to fixation of the arytenoid by post-cricoid or hypopharyngeal neoplasm have not been included

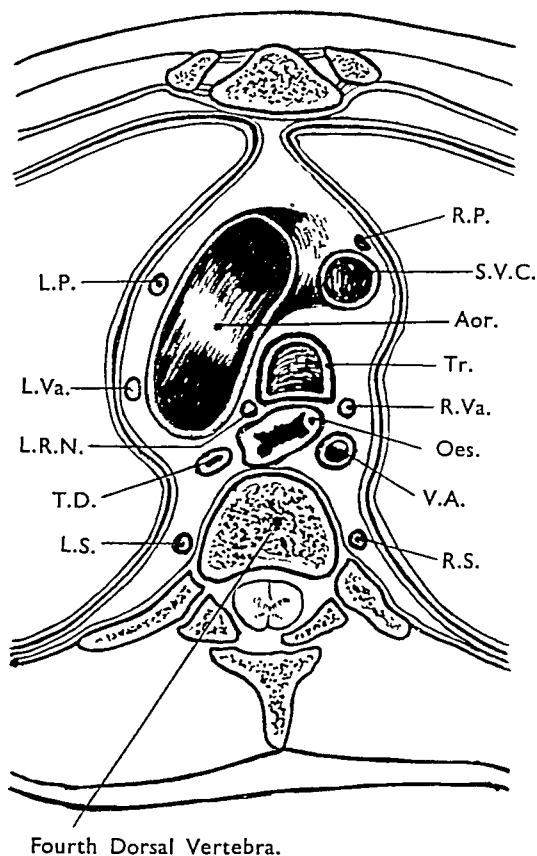


FIG. 70.—Relations of œsophagus at level of fourth dorsal vertebra. L.P. and R.P., Phrenic nerves; L.R.N., Left recurrent nerve; R. and L. Va., Vagi; R.S. and L.S., Sympathetic nerves; T.D., Thoracic duct; V.A., Azygos vein.

in this series. The paralysed cords when viewed by the laryngeal mirror were seen to be in the cadaveric position. In one patient with left-cord paralysis who later developed a right-cord palsy, it was possible to observe the sequence of abductor paralysis followed by the adoption of the cadaveric attitude.

The four patients who exhibited right-cord palsy were all males with neoplasms situated respectively at 11 in., 13 in., 13½ in., and 14 in. from the incisor teeth.

The growths in these four cases were far below the neighbourhood of the right recurrent nerve, and the paralyses were probably due to pressure on the nerve by enlarged lymphatic glands of the right paratracheal (recurrent) chain (*see Fig. 75*); consequently these cases are included under the heading of lymphatic spread, leaving 9 cases only which clinically showed evidence of recurrent nerve involvement due to direct spread from an Œsophageal neoplasm.

Table II.—PERCENTAGE LOCATION OF ŒSOPHAGEAL CARCINOMA

OBSERVER	UPPER THIRD	MIDDLE THIRD	LOWER THIRD
Clayton ¹² ..	Per cent 10	Per cent 56	Per cent 34
Kraus ¹⁷	18	36	46
Janeway and Green ¹⁷ ..	16	32	52
Watson ¹	19	27	54
Guisez ¹⁸	34	46	20
Mackenzie ¹⁸ ..	13	50	37
Rawling ¹⁸ ..	10	80	10
Binnie ¹⁸	16	32	52
Lamy ¹⁸	10	36	54
Abel ⁷	29	46	25
Souttar ⁵	22	63	15
Cleminson and Monkhouse ¹⁹ ..	7	62	31
Klein ²⁰	14·6	46·7	27·2
Von Hacker ²⁰ ..	12·3	48·8	26
Lotheissen ²⁰ ..	13·3	48·8	37
Kraus-Ridder ²⁰ ..	27	30	56
Present series ..	22	51	27
Mean percentage	17	47	36

The patient who exhibited bilateral vocal-cord palsy was the only one who gave a history of hoarseness *preceding* dysphagia:—

Case 1.—W. A. B., male, aged 60 years. Complained of hoarseness of four months' and dyspnœa of one month's duration. He stated that dysphagia had been present for fourteen days only. Examination showed that both vocal cords were fixed in the cadaveric position. Probably his left cord had become paralysed four months before coming to hospital, giving rise to hoarseness, and this was followed three months later by paralysis of the right cord, which caused the dyspnœa. This was an interesting case, as he had two malignant ulcers of the Œsophagus, which are described and illustrated below (*see Fig. 76*).

Recurrent-nerve paralysis indicates extensive extra-oesophageal spread, which should definitely exclude any attempt at radical treatment. Only one patient of the present series with left-cord paralysis came to autopsy, and examination showed a small ulcer of the middle third of the oesophagus, whilst the entire mediastinum was filled with carcinomatous growth apparently invading all the neighbouring structures. Guisez,²² from his wide experience, regards such cases as unsuitable even for palliation by radium, and he has stated²³ that 1 out of 5 of all cases of carcinoma of the oesophagus develop vocal-cord paralysis, and that when the growth is situated in the upper third paralysis occurs in 1 out of every 2 cases. He records 60 cases, of which 6 were bilateral. New and Chidley²⁴ have reported 34 cases of recurrent-nerve paralysis in patients suffering from carcinoma of the oesophagus. In their series the left cord was affected in 18 cases, the right in 13, and in 3 the paralysis was bilateral. Watson,¹ in his large series of 245 cases, reports 'hoarseness' as a symptom in 2 only, and Clayton¹² states that of a series of 41 autopsies, hoarseness had been complained of in 5 cases.

Phrenic Nerve.—It has been stated by several observers that a troublesome hiccup occurring in the course of carcinoma of the oesophagus is indicative of involvement of the phrenic nerve—this would probably be due to an irritative lesion. Abel⁷ states that paralysis of the diaphragm and massive collapse of the lung may occur as secondary phenomena in the presence of phrenic nerve paralysis. Hiccup was the first symptom complained of by one patient of the present series:—

Case 2.—W. B. S., male, aged 62 years. Complained in June, 1934, of persistent hiccup, some giddiness, and nose bleeding; he made no complaint of dysphagia. His blood-pressure was 205/125, and he had some cardiac hypertrophy and arteriosclerosis. In August, following routine X-ray examination, Dr. J. H. Mather reported: 'The appearances are typical of early malignant disease of the oesophagus at level of 8th D.V. There is a slight hold-up of thick paste at the site of the growth, but fluids pass without difficulty.' Œsophagoscopy showed a small malignant ulcer 10½ in. from the incisor teeth into which radon seeds were inserted. A snipping removed was reported by Dr. Howel Evans to be epitheliomatous. This was considered to be an early and fairly hopeful lesion, and the presence of extra-oesophageal spread was not anticipated as the significance of the hiccup was not recognized. The patient died five months later of auricular fibrillation. No autopsy was obtained.

Watson¹ records two cases of severe hiccup in his series.

Intercostal Nerves and Vertebrae.—The intercostal nerves are not infrequently affected either directly or as a sequel of carcinomatous extension of the vertebrae. Carcinoma of the oesophagus is essentially a painless disease, and when pain is present it is an indication of extra-oesophageal spread. Discomfort and fullness behind the sternum are common complaints, and odynphagia may be present in growths of the extreme upper end of the oesophagus, but pain occurring independently of swallowing in patients with tumours situated below this level is a symptom of serious import. Four patients of the present series sought advice because of severe pain—they complained also of dysphagia, but in all four cases this was a minor complaint.

Case 3.—A. B., female, aged 60 years. With a malignant ulcer in the upper third of her oesophagus complained of severe pain from which she had suffered for three months. X-ray examination showed a malignant stricture of the oesophagus with definite destruction of the body of the seventh cervical vertebra (*Fig. 71*).

Two male patients, aged 57 years and 63 years respectively, with middle-third growths experienced agonizing girdle pain. Both of these patients could swallow

comfortably after intubation, but it was found necessary to keep them under the influence of large doses of morphia to relieve their suffering. X-ray films of one of these patients showed involvement of the body of the eighth dorsal vertebra; no X-ray changes in the spinal column were manifest in the other patient.

The fourth case was that of a female patient suffering from carcinoma of the upper œsophagus with extension to the thyroid gland. She complained of constant and severe pain aggravated by attempts to swallow—*Case 4* described below.



FIG. 71.—*Case 3 A*, Carcinomatous destruction of seventh cervical vertebra.

Sympathetic Nerve.—Sympathetic-nerve involvement was not observed in the present series. Abel⁷ and Guisez²² both call attention to the symptoms of such a complication—an early irritative lesion followed by paralysis with its characteristic unilateral signs (Horner's syndrome).

Thyroid Gland.—Direct spread of upper œsophageal growths into the thyroid gland does occur, and one case was noted in the present series:—

Case 4.—A. L., female, aged 54 years. Stated that she had experienced severe pain in her throat and difficulty in swallowing for the past four months, and that one month ago she had noticed a swelling of the left side of her neck. Examination showed a hard fixed enlargement of the left lobe of the thyroid gland, and paralysis of the left vocal cord. Endoscopy revealed an extensive carcinoma of the upper œsophagus involving the trachea and thyroid gland. The patient became very dyspnoëic, and an emergency tracheotomy was necessary one week after her first application to hospital.

Respiratory Tract.—Involvement of the trachea, the bronchi, pleuræ, or lungs is probably the next most common complication observed during life after recurrent-nerve involvement, and it is in a large proportion of cases the immediate

cause of death. Souttar⁵ has stated that 'of every 10 cases of carcinoma of the œsophagus' 9 will die from purulent bronchopneumonia and 1 from hæmorrhage. In Watson's¹ series 48 per cent of the cases coming to autopsy had died from bronchopneumonia.

Keefer²⁵ has reported a series of 17 cases of carcinoma of the œsophagus in which the dominant symptoms were pulmonary, and he has tabulated the causes of such respiratory complications as follows:—

1. Aspiration owing to œsophageal obstruction.
2. Non-perforating involvement of the trachea, bronchus, pleura, or lung.
3. Tracheo-bronchial stenosis due to (a) Direct spread of growth, or (b) Pressure by metastatic glands.
4. Perforation by growth into trachea, bronchus, pleura, or lung.
5. Perforation by metastatic glands into trachea, bronchus, pleura, or lung.

Such lesions are characterized by cough, hæmoptysis, and dyspnœa.

Three cases of the present series showed a non-perforating lesion of the trachea at the time of the first examination:—

Case 5.—F. C., male, aged 61 years. Complained of dysphagia of three months' duration and of coughing when lying in bed. He gave a history of syphilis thirty years previously, but his blood Wassermann reaction was negative. X-ray report: 'Marked dilatation of the upper portion of the œsophagus. The X-ray appearances point to a malignant stricture rather than the presence of a pouch.' Œsophagoscopy showed a hard, ulcerated stricture 10½ in. from the incisor teeth, which was very difficult to dilate for intubation as bleeding was very profuse. Tracheoscopy revealed a granulating mass of growth on the posterior tracheal wall situated immediately above the bifurcation (*Fig. 72*). The patient died one month later, and the post-mortem report was as follows: 'Right-sided pneumothorax—pleural cavity contained about two pints of fluid. Œsophagus—Souttar's tube in position. Œsophageal growth around the wall has ulcerated into trachea—no perforation. Numerous secondary glands. Liver normal.'



FIG. 72.—*Case 5.* Non-perforating involvement of the trachea from œsophageal carcinoma.

Case 6.—W. T. M., male, aged 59 years. Complained of difficulty in swallowing of two months' duration, and more recently of nocturnal cough. X-ray examination showed 'malignant disease of the œsophagus at level of fourth to seventh dorsal vertebræ.' The upper margin of an œsophageal neoplasm was found by œsophagoscopy at 9½ in. from the incisor teeth. Tracheoscopy revealed a similar lesion to that observed in *Case 5*.

The third case is reported above (*Case 4*).

It is of interest to note that *Cases 5* and *6* both complained of a cough which was troublesome when in the supine position. A third patient also complained of a troublesome cough at night. He was treated with radon seeds two years ago for malignant disease of the upper third of his œsophagus and is still alive. He was examined recently and it was found that he had paralysis of his left vocal cord and a cough which interfered with his sleep at night, but was not troublesome during the day. In view of the two cases reported above, it is presumed that he, also, has a non-perforating extension to his trachea.

Nocturnal cough as a symptom of non-perforating tracheal involvement does not appear to have been described elsewhere, and it is possibly due to irritation caused by the backward pressure of the trachea on to the ulcerating mass when the patient adopts the supine position.

Souttar²⁶ has recorded one case in which a nodule of growth in the trachea spreading from an œsophageal neoplasm actually caused respiratory embarrassment, and one case of the present series (*Case 4*) required tracheotomy owing to tracheal stenosis due to a non-perforating involvement of the trachea by direct spread.

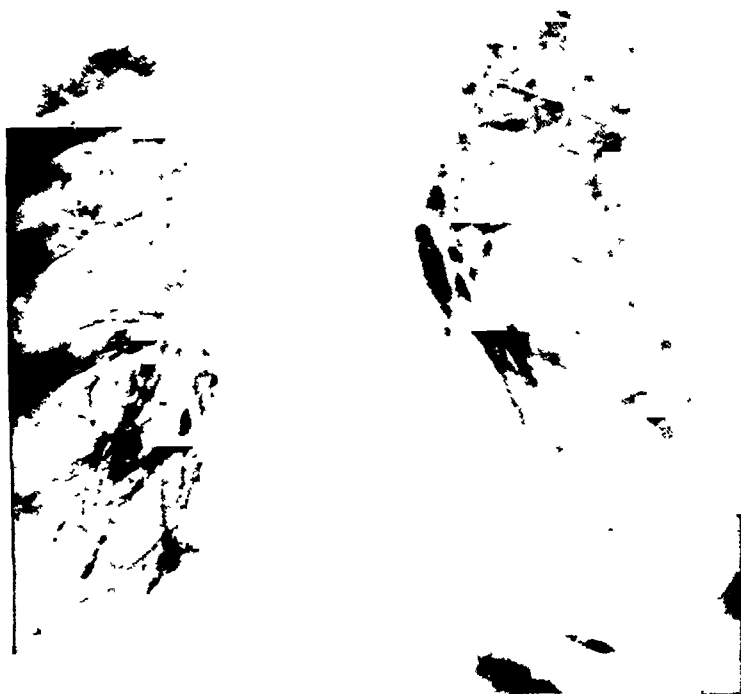


FIG. 73 —Barium paste in the bronchi after swallowing in a patient with a carcinomatous œsophago-tracheal fistula

Perforation of the trachea or bronchi is characterized by a cough which occurs in violent paroxysms and suffocative spasms during attempts to swallow, and this is usually accompanied by fœtid expectoration. In such cases, if the patients can take barium paste, it will be seen to pass into the trachea and bronchi, partially outlining the bronchial tree (*Fig. 73*). Kerley²⁷ has described how he watched such a happening under the fluorescent screen in a patient with carcinoma of the middle third of the œsophagus. The barium passed into the trachea and down to the bases of the lungs, and the patient became very distressed and dyspnoëic; half an hour afterwards he was quite well, and the examination was repeated some time later with similar results. No lung complications ensued, and the patient did not die until some months later, and then owing to cachexia and exhaustion.

There was only one patient in the present series who developed a tracheal fistula after diagnosis of the œsophageal lesion was made, and it was impossible to X-ray him, as the ingestion of the smallest amount of fluid caused a severe paroxysm of coughing and great distress :—

Case 7.—V. R., male, aged 62 years. Complained of dysphagia of three months' duration. X-ray and œsophagoscopy showed a malignant stricture 11 in. from the incisor teeth. This was dilated and treated with radon seeds. One month later he returned to hospital complaining of inability to swallow owing to 'choking' and of putrid purulent expectoration. He died six and a half weeks later, and autopsy showed a large perforation which admitted two fingers from the anterior wall of the œsophagus into the posterior wall of the trachea (*Fig. 74*).

Such a complication is an urgent indication for gastrostomy.

Guisez²⁸ has stated that he has observed tracheal involvement in 15 per cent of his cases, and he quotes Zenker and Ziemssen's series of 120 cases with 26 perforations into the left bronchus and 20 into the trachea. Scott-Brown²⁹ described 4 cases of fistula into the air-passages; he recorded that one of these patients lived for seven months after the diagnosis of perforation was made, and Grey Turner³⁷ has reported the case of a man who lived for nine months with an œsophago-tracheal fistula. Watson¹ has stated that of his series of 27 post-mortem examinations 7 cases showed extension to or actual rupture into the trachea or bronchus; and Souttar⁵ has reported 3 cases with perforation into the left bronchus and 3 into the trachea itself of his series of 18 autopsies.



FIG 74—*Case 7* A, Perforation from œsophagus into trachea

Involvement of the pleuræ and lungs frequently becomes manifest in the terminal phases of carcinoma of the œsophagus, and this complication is commonly observed at autopsy. When pleural pain occurs it is usually a *very* late symptom. Clayton¹² has reported 6 cases who had complained of such pain out of a series of 41 autopsies.

One patient of the present series complained of pleural pain at her first visit to hospital :—

Case 8.—S. B., female, aged 52 years. Complained of dysphagia of four months' duration and that latterly she had suffered from pain in her right chest. X rays showed irregularity of the barium shadow from the level of the seventh dorsal vertebra to the eleventh dorsal vertebra with dilatation above this region. A few days later an effusion into her right pleural cavity became evident. She was admitted to hospital, where she died three weeks later. The post-mortem examination report was as follows: 'In the lower third of the œsophagus and extending for 4 in. is a large epitheliomatous ulcer; the growth almost blocks the lumen. Its lower extremity extends to 1 in. of the cardiac orifice. The right pleural cavity is filled with purulent fluid. Leak in the cancerous wall of the œsophagus. Mass of glands in neighbourhood of head of pancreas.'

LYMPHATIC SPREAD

The œsophagus is richly supplied with lymphatics, which are present as two networks—a submucous and a muscular. These are continuous with similar

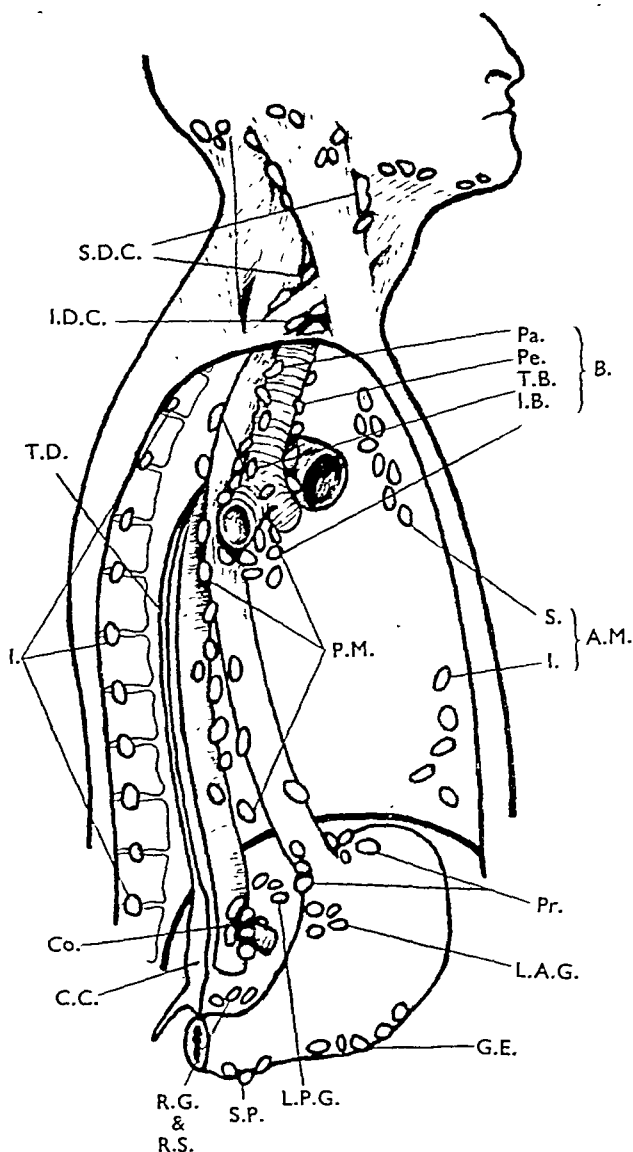


FIG. 75.—Lymphatic drainage of the œsophagus. S.D.C. and I.D.C., Superior and inferior deep cervical; Pa., Paratracheal; Pe., Pretracheal; T.B., Tracheo-bronchial; I.B., Interbronchial; B., Bronchial; S. and I.A.M., Superior and inferior anterior mediastinal; P.M., Posterior mediastinal; I., Intercostal; T.D., Thoracic duct; C.C., Cisterna chyli; Co., Coeliac; Pr., Paracardial; L.A.G., Left anterior gastric; G.E., Gastro-epiploic; L.P.G., Left posterior gastric; S.P., Subpyloric; R.G., Right gastro-epiploic; R.S., Right suprapancreatic.

lymphatic systems of the pharynx above and the stomach below. The efferents from these networks drain into the neighbouring lymphatic glands (*Fig. 75*). Those from the upper half drain into the posterior mediastinal, the deep cervical, and paratracheal glands: those from the lower half drain into the posterior mediastinal, paracardial, left anterior, and posterior gastric glands, and thence into the celiac glands. There is hardly any limit to their anastomoses in the neck, thorax, or abdomen, as obstruction in any one gland may lead to reversal of lymph-flow and opening up of new paths. Musgrave Woodman⁹ has injected experimentally the



FIG. 76.—Case 1.—Radiogram showing two malignant strictures of the œsophagus

œsophageal lymphatics of monkeys with methylene blue. The monkeys were killed twelve days later, and examination showed widespread diffusion of the dye into the lymphatic system, including the glands at the base of the neck, the mediastinal and intercostal glands, and a remarkable extension to the parietal pleura and the pleural surface of the diaphragm. The inference he draws from his experiments is that lymphatic spread in the early stages of carcinoma is slow and takes place with difficulty, but when once the fine reticulum has been broken through the invasion is widespread and comparatively rapid.

Permeation of the local mural lymphatics may take place, and off-shoots from these to the surface of the œsophagus may give rise to apparently multiple new growths with gaps of normal mucous membrane separating the neoplastic areas. Although this method of spread is often seen at autopsy, it is rarely encountered during life—probably because it is difficult and dangerous to pass an œsophagoscope through a malignant stricture and so diagnose a growth lower down. Guisez³⁰ has described two cases of multiple ulcers of the œsophagus seen during life—one case with three ulcers, and one with two. Chevalier Jackson² saw multiple ulcers twice only in a series of 671 cases. Souttar³¹ states that these neoplastic upshoots may give rise to multiple strictures of the œsophagus, and an illustration of this complication is reproduced in *Fig. 76*. Four cases of multiple œsophageal ulcers occurred in the present series, two of which have been reported elsewhere.³² Briefly they were as follows:—

Case 9.—G. B., female, aged 46 years. Gave a history of four months' dysphagia. X rays, œsophagoscopy, and biopsy showed an annular carcinomatous ulcer 10 in. from the incisor teeth. Radon seeds were inserted. Three months later she returned with a malignant stricture situated at 12 in. from the incisor teeth, the original ulcer being healed as a fibrous stricture.

Case 10.—J. O., male, aged 65 years. Complained of dysphagia of nine months' duration. Œsophagoscopy showed ulcers situated at 7½ in and 11½ in. from the incisor teeth. Snippings from both ulcers were reported to be epitheliomatous by Professor Beattie.

Case 1.—W. A. B., male, aged 60 years. Came to hospital suffering from bilateral recurrent nerve paralysis, and his symptoms have been described above. He was very emaciated; and X-ray examination by Dr. Whitaker demonstrated two strictures of the œsophagus (*Fig. 76*). Œsophagoscopy showed a malignant stricture at the entrance of the œsophagus and another situated at 13 in. from the incisor teeth. Biopsy proved both growths to be epitheliomatous.

Case 11.—R. W., male, aged 62 years. Complained of dysphagia of eight weeks' duration. X rays showed a 'stricture, probably malignant, of upper third of the œsophagus.' Œsophagoscopy demonstrated a submucous non-ulcerated growth 8½ in. from the incisors and a large annular malignant stricture at the lower end of the œsophagus. This patient died four months later and autopsy showed a small growth of the upper third of the œsophagus just beginning to ulcerate, and a large ulcerated growth of the lower third (*Fig. 77*). Many of the mediastinal structures were involved by extra-œsophageal growth—pleuræ, pericardium, etc.—and there were numerous glandular secondaries in the celiac lymphatic group.



FIG. 77.—*Case 11.* A, Superior ulcer; B, Inferior ulcer.

Multiple strictures may also be caused by pressure of enlarged metastatic glands. Keefer²⁵ has reported such a case. Œsophageal obstruction due to a secondary glandular mass occurred in one case of the present series (*Case 13*) which is reported later. This was observed only at autopsy and was not diagnosed during life.

There can be no doubt that lymphatic glandular deposits and lymphatic extension to more distant organs is commonly seen at autopsy—yet the question of the frequency of secondary lymphatic involvement is unsettled and is fully discussed by Abel,⁷ Souttar,⁵ and Watson.¹ Only 13 cases of the present series have come to autopsy, and of these 10 showed macroscopic lymphatic-gland involvement in the thorax and/or in the abdomen. The presence of metastatic glands is not often recognized during life, as the patients usually succumb before recognizable symptoms attributable to glandular enlargement become manifest. Souttar²⁶ has stated that masses of enlarged glands may become manifest in the neck or epigastrium of patients suffering from carcinoma of the Œsophagus, and Chevalier Jackson² recorded that out of a series of 671 cases in 16 the Œsophageal lesion gave rise to no symptoms and was only discovered in seeking for a cause of cervical adenopathy.

Five cases occurred in the present series :—

Case 12.—F. E., female, aged 53 years. Referred from Mr. W. R. Williams's Clinic for investigation, with enlarged palpable glands in her left supraclavicular triangle. The enlargement was of six weeks' duration, and the patient made no complaint of dysphagia until questioned. X rays showed some delay of opaque paste at the upper end of the Œsophagus, suggestive of a post-cricoid carcinoma. Œsophagoscopy revealed a malignant ulcer of the Œsophagus immediately below the cricoid.

The remaining four cases have already been mentioned and were males suffering from right recurrent-nerve palsy. In all four cases the growths were situated in the lower third of the Œsophagus, and it is reasonable to assume that the nerve lesions were due to pressure or involvement by glandular metastases.

BLOOD-STREAM SPREAD

Venous spread is probably rare. Abel⁴ has recounted the findings of Leichenstein, who observed a case in which direct extension took place along the azygos vein to the superior vena cava and thence to the right auricle and right ventricle; and Souttar⁵ has recorded the post-mortem findings in a male patient aged 62 years who exhibited no signs of carcinoma of the Œsophagus during life, but at autopsy a large Œsophageal carcinoma was found with metastases in the cerebellum.

One case of the present series is probably an example of blood-borne spread :—

Case 13.—M. A. B., female, aged 54 years. Stated that she had pain in her chest for two months when swallowing. X rays, Œsophagoscopy, and biopsy showed a malignant ulcer of her Œsophagus at 9 in. from the incisor teeth. This was treated with radium. A month later she complained of a lump on her right hip, and she entered another hospital, where she died seven months later. I am indebted to Professor T. B. Davie for the following post-mortem report: 'Thorax: large secondary mass growing in eleventh rib right up to vertebral column—pleural surface not involved. Lungs: one secondary mass near left apex involving lung tissue but possibly only secondarily from a gland metastasis. This mass is adherent to the outside of the Œsophagus and is visible from within the Œsophageal lumen as a protuberance just above the tracheal bifurcation. Œsophagus: no present ulceration anywhere. At level of tracheal bifurcation the lumen is slightly obstructed by protuberance inward over adherent gland mass—no definite scar within the Œsophagus. Abdomen: one gland mass

on lesser curvature just below diaphragm. No growth in stomach. Liver, kidneys, and spleen : all normal. Adrenals : left involved in malignant metastases. Bones : large metastasis on surface of right iliac bone eroding bone substance.'

SPREAD BY IMPLANTATION

Spread by implantation is rare, and when this does occur in the œsophagus itself it is impossible to state definitely without a careful microscopical examination that the secondary lesion is not due to spread by the deep lymphatics as described above.

Two cases of undoubted implantation spread occurred in the present series, and a third case is described which appeared to be an example of endo-œsophageal spread by this method. One of the former cases has been recorded elsewhere.³²



FIG. 78.—Case 15 Multiple oral neoplastic ulcers associated with carcinoma of the œsophagus.

Case 14.—R. K., male, aged 62 years. Was treated at the Royal Southern Hospital with radium in November, 1932, for a carcinomatous stricture of the œsophagus at $13\frac{1}{2}$ in. from the incisor teeth. In February, 1933, radium was applied to a carcinoma of the left side of his tongue by Mr. R. Kennon at the Liverpool Royal Infirmary. He died in August, 1933, with numerous metastases from the lingual and œsophageal primaries.

The second case was seen more recently by the courtesy of Mr. W. R. Williams :—

Case 15.—H. H., male, aged 62 years. Stated that four months previously he first noticed some difficulty when swallowing. Two months later he had suffered from bronchitis and pleurisy, since when swallowing had been more difficult, and he could only take thick liquids at the time of his admission to hospital. A lump had appeared in the left side of his neck two months ago and on the right side six weeks ago—neither of which was painful. His throat had been sore for the past three weeks. Examination showed a large neoplastic ulcer involving the right anterior faucial pillar, hard palate, and buccal surface of the right cheek ;

and a smaller ulcer of the buccal mucosa situated at the region of the junction of the ascending and horizontal rami of the mandible (*Fig. 78*). There were hard fixed masses of inoperable glands situated in both anterior triangles. X-ray examination of the œsophagus disclosed a 'hold-up of barium at level of seventh dorsal vertebra due to malignant stricture of œsophagus—? specific.' Blood Wassermann reaction was negative. Œsophagoscopy revealed an extensive annular carcinomatous stricture of the œsophagus 13 in. from the incisor teeth. Snippings from both faucial ulcers and from the œsophageal growth were reported by Dr. R. Mole to be epitheliomatous. A Souttar's tube was inserted through the stricture, but the patient died two months later. No autopsy was obtained.

The third case exhibited what appeared to be an implantation growth in the œsophagus itself—this was a presumptive diagnosis only, as it was impossible to prove that the spread was not due to lymphatic permeation:—

Case 16.—W. P., male, aged 62 years.

Complained of a 'feeling of something in his throat' when swallowing of four weeks' duration. X rays demonstrated a partial hold-up immediately below the cricoid. Œsophagoscopy showed a proliferating ulcer of the anterior wall of the œsophagus $7\frac{1}{2}$ in. from the incisor teeth. Immediately opposite to this on the posterior wall was a small solitary nodule of growth (*Fig. 79*). The mucous membrane separating the growths was normal. There appeared to be no infiltration of these areas, and the nodule suggested a surface infection—a true implantation growth.



FIG. 79.—*Case 16.* Multiple growths of œsophagus.

The two former cases are very unusual, and I can find only one reference to proved implantation secondary growths from an œsophageal carcinoma. Hoche³³ described a case in which an epithelioma arose on a gastric ulcer in a patient suffering from carcinoma of the œsophagus.

CACHEXIA

Five patients of the present series arrived at hospital in an advanced state of starvation and exhaustion. All were considered to be unfit for palliative treatment, even by dilatation or intubation. Four cases were admitted forthwith and treated with sedatives and proctoclysis. The fifth case was sent home to await admission, but he was found dead in bed on the following day.

SURGICAL REMOVAL

Only one patient of the present series of 100 cases was considered suitable for an attempt at excision, but operation proved the growth to be too extensive for removal. The case was examined by the kindness of Mr. C. A. Wells, who subsequently operated:—

Case 17.—P. H., male, aged 44 years, gave a history of four months' dysphagia. X-ray report: 'Hold-up at extreme upper end of œsophagus.' Œsophagoscopy showed the upper edge of a proliferating neoplasm 1 in. below the cricoid. The visible growth appeared to be situated on the right lateral wall and suggested a comparatively localized lesion. The

œsophagoscope was not passed through the neoplastic area. A gastrostomy was performed by Mr. Wells, who later exposed the upper œsophagus through a right cervical incision, intending to use a Trotter's skin-flap. The growth was found to be very extensive, passing down for some inches into the mediastinum and adhering to and infiltrating all the neighbouring prevertebral structures. The operation was abandoned and two days later a leak from the œsophagus into the neck wound became manifest. The patient died within a short period. No autopsy was performed.

COMMENTARY

The case histories of 100 cases of carcinoma of the œsophagus have been examined in order to ascertain the reason for the non-performance of radical surgical treatment.

Of this series in one case excision of the tumour was contemplated, but operation proved the lesion to be too extensive for removal. Severe cachexia and exhaustion was shown by five patients on their arrival at hospital so that simple alleviation only of their suffering until death was possible. In 65 cases X rays and œsophagoscopy revealed so extensive a local lesion that palliative treatment alone was considered to be feasible. In 29 cases evidence of spread of the growth was present at or about the time of the first application for treatment.

This latter figure is a surprisingly high one, and the findings bear witness to the tragedy of late diagnosis. The loss in this manner of valuable time is due largely to lack of appreciation by the general public of the gravity of any abnormality of the act of swallowing, and of the necessity at the first occurrence of such complaint for a complete clinical, radiographic, œsophagoscopic, and bronchoscopic investigation. This will certainly involve the performance of many examinations with negative results, but it is the only routine by which at present the early case can be discovered. No investigation of a suspected case of œsophageal neoplasm is complete without direct inspection of the trachea and larger bronchi, and this procedure should be carried out at whatever level the œsophageal lesion is situated, as not only may the primary growth involve any portion of the lower respiratory tract, but such a complication may be due to invasion from metastases.

The course of carcinoma of the œsophagus can be divided clinically into three phases :—

1. The latent period—which is symptomless and of unknown duration.
2. The 'symptom' period—i.e., the time lost between the incidence of the earliest symptoms and the date of a definite diagnosis. This period averages 5·7 months.
3. The 'manifest' period, i.e., the survival time of the patient from the date of diagnosis until death. This period averages 5·14 months.

Chevalier Jackson,² who has tabulated the early symptoms of carcinoma of the œsophagus, is of the opinion—with which all those who are interested in the subject will agree—that in medical education the importance of the so-called late symptoms should be minimized, whereas the description of the early symptoms, which has not yet found its way into text-books, should be emphasized. He has further stated³⁴ that 'not until every patient who complains of cervical, retro-sternal or epigastric abnormal sensation is regarded *not* as neurotic but as possibly cancerous, will the surgeon have a chance.'

The results of the above analysis are not submitted as a discouragement to

attempts at radical surgical methods. That such methods are firmly established on a secure technique is evidenced by the brilliant achievements of British and Continental surgeons—of the former Professor Grey Turner³⁵ and Mr. King³⁶ recently have reported successful cases of removal of the entire thoracic œsophagus. The findings, however, would indicate that if diagnosis was made at the onset of the 'symptom' period, then Abel's contention that at least one case out of every four could be cured by radical surgical methods might be tenable.

In addition to the acknowledgements made in the text, I am indebted to my colleagues at the Royal Southern Hospital for permission to examine and treat their cases, and particularly are my thanks due to Drs. J. H. Mather, R. E. Roberts, P. Whitaker, and A. E. Connolly for numerous expert X-ray examinations without which it would have been impossible to record these notes.

REFERENCES

- ¹ WATSON, W. L., *Surg. Gynecol. and Obst.*, 1933, lvi, 884.
- ² JACKSON, CHEVALIER, *Amer. Jour. Med. Sci.*, 1925, clxix, 625.
- ³ JACKSON, CHEVALIER, *Bronchoscopy and Œsophagoscopy*, 1927, 296.
- ⁴ ABEL, A. L., *Brit. Jour. Surg.*, 1926, xiv, 131.
- ⁵ SOUTTAR, H. S., *Ibid.*, 1927, xv, 71.
- ⁶ BRODERS, A. C., and VINSON, P. P., *Arch. Otol.*, 1928, viii, 79.
- ⁷ ABEL, A. L., *Œsophageal Obstruction*, 1927, 168.
- ⁸ WRIGHT, A. J., *Brit. Jour. Surg.*, 1927, xv, 71.
- ⁹ WOODMAN, E. MUSGRAVE, *Brit. Med. Jour.*, 1931, ii, 290.
- ¹⁰ VINSON, P. P., *Proc. Staff Meeting Mayo Clinic*, 1931, June 14.
- ¹¹ ROBERTS, O. W., *Brit. Jour. Surg.*, 1930, xvii, 683.
- ¹² CLAYTON, E. S., *Surg. Gynecol. and Obst.*, 1928, xlvii, 52.
- ¹³ HELSLEY, F. G., *Ann. of Surg.*, 1928, lxxvii, 272.
- ¹⁴ EWING, J., *Neoplastic Disease*, 1928, 904.
- ¹⁵ ILLINGWORTH, C. F. W., and DICK, B. M., *Textbook of Pathology*, 1932, 409.
- ¹⁶ LOWE, E. CRONIN, Personal communication.
- ¹⁷ Quoted by Watson.¹
- ¹⁸ Quoted by Abel.⁷
- ¹⁹ CLEMINSON, F. J., and MONKHOUSE, J. P., *Jour. Laryngol. and Otol.*, 1934, xlix, 313.
- ²⁰ GRAHAM, E. A., and BALLON, H. C., *Ann. of Otol. Rhinol. and Laryngol.*, 1931, xl, 896.
- ²¹ SEMON, SIR F., *Arch. Otolaryngol.*, 1881, ii, 197.
- ²² GUISEZ, J., *Bull et Mém. Soc. de Chir.*, 1930, xxii, 751.
- ²³ GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923.
- ²⁴ NEW, G. B., and CHIDLEY, J. H., *Arch. Otolaryngol.*, 1932, xvi, 143.
- ²⁵ KEEFER, C. S., *Ann. of Internal Med.*, 1934, viii, 72.
- ²⁶ SOUTTAR, H. S., *Brit. Med. Jour.*, 1934, ii, 798.
- ²⁷ KERLEY, P., *Recent Advances in Radiology*, 1931, 195.
- ²⁸ GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923, 139.
- ²⁹ SCOTT-BROWN, W. G., *Lancet*, 1934, ii, 544.
- ³⁰ GUISEZ, J., *Rétrécissements de l'Œsophage*, 1923, 154.
- ³¹ SOUTTAR, H. S., *Choyce's System of Surgery*, 1932, 184.
- ³² MCGIBBON, J. E. G., *Lancet*, 1933, ii, 806.
- ³³ HOICHE, L., *Presse méd.*, 1901, i, 67.
- ³⁴ JACKSON, CHEVALIER, *Ann. Otol. Rhinol. and Laryngol.*, 1931, xl, 889.
- ³⁵ TURNER, G. GREY, *Proc. Roy. Soc. Med.*, 1934, xxvii, 355.
- ³⁶ KING, E. S. J., *Brit. Jour. Surg.*, 1936, xxiii, 521.
- ³⁷ TURNER, G. GREY, *Lancet*, 1936, i, 68.

INTRAVENOUS INJECTION OF HYPERTONIC SODIUM CHLORIDE SOLUTION IN THE TREATMENT OF SOME CONDITIONS OF LOW BLOOD-PRESSURE

BY JOAN B. WALKER, LEICESTER

HYPERTONIC sodium chloride solution has been used intravenously for several different purposes. It has been given to reduce an increased intracranial pressure from œdema or hæmorrhage;¹ to promote intestinal motility in cases of paralytic ileus;² and in cholera³ it has been given in order to attract water to the blood, which is viscous owing to extreme dehydration of the body. It has not been in general use for the purpose of raising a lowered blood-pressure, although it has been suggested by Graham⁴ that a pint of 1·2 per cent sodium chloride should be given to raise the blood-pressure in diabetic coma, as in this condition the tissues lack water and chlorine, and sodium chloride also helps to neutralize acetone bodies. Other workers have put forward objections to the use of hypertonic solutions in the treatment of shock. Bayliss,⁵ in his Oliver Sharpey Lectures of 1918, reviewed the physiological and therapeutic action of various solutions that could be employed by intravenous injection for the treatment of wound shock. He came to the conclusion that a hypertonic solution of sodium chloride was inadvisable as he found the effect transitory and he had noticed unpleasant symptoms of gasping respiration and slowing of the heart. He stated that he believed it undesirable to add to the saline content of the organism, which is an interesting observation when considering the recent treatment of Addison's disease, with its associated low blood-pressure, by sodium chloride.^{6,7} If it is undesirable to add to, it seems essential to maintain, the balance of circulating sodium chloride, both on account of its chemical and of its physical properties.

MacFee and Baldrige,⁸ also when considering the infusion treatment of shock, state that they 'see small logic in the use of hypertonic solutions', because of the temporary hydræmia, rapid excretion by the kidneys, and finally a loss rather than gain of fluid.

Cushing and Foley⁹ find that the raised blood-pressure may be an objection to the use of hypertonic saline intravenously for reducing intracranial tension.

With due regard for the objections mentioned above, the present series of observations were made on 53 cases. In 49 of these hypertonic saline was injected intravenously with the object of raising a lowered blood-pressure before, during, or after operation. Most cases received 40 c.c. of a 30 per cent solution (12 grm.) of sodium chloride. Blood-pressure records were kept which show the rapidity and duration of the reaction.

The two cases which suggested the possibility of using small quantities of strongly hypertonic salt solution for its rapid stimulating effect on the circulation happened to occur on the same day. In the first case 30 c.c. of 30 per cent sodium chloride solution were injected intravenously, with the aim of reducing intracranial

tension to close the dura following an exploratory operation for cerebral tumour. A record had been kept for over two hours and the systolic blood-pressure had been between 140 and 130 mm. Hg. Within five minutes of the injection it rose from 130 to 165, but the effect was transitory.

Fig. 80 shows the record of the second case—that of a woman aged 60. Spinal anaesthesia was administered for laparotomy. Paralytic ileus due to pneumococcal peritonitis was subsequently found. There was severe initial shock; the systolic blood-pressure fell from 140 to 70 mm. Hg: 40 c.c. of 30 per cent NaCl solution were injected intravenously. This time, with the morning's case in mind, it was hoped that there would be the same quick rise in blood-pressure; also there was

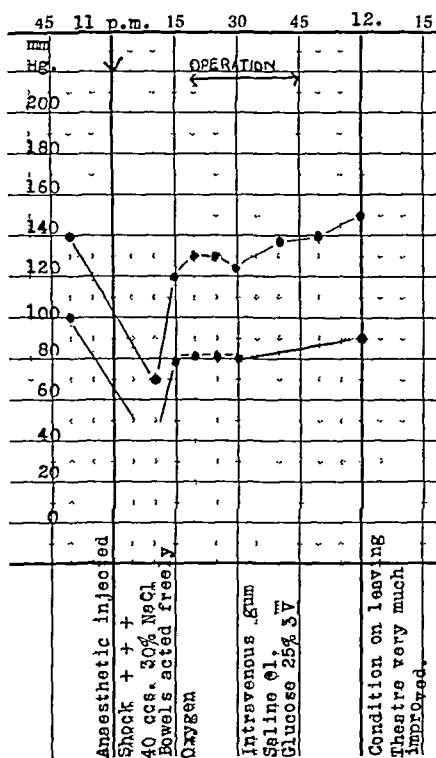


FIG 80—Case 2. Female, aged 60. Pneumococcal peritonitis

the idea of possibly replacing the chlorides in the blood lost by her repeated vomiting, and of promoting peristalsis. It was to be followed by a pint of gum saline and glucose. Five minutes after the injection the blood-pressure rose from a systolic reading of 70 to 120. A copious evacuation of the bowels occurred, the operation was performed, and the patient returned to bed in an improved condition.

The improvement in this case was sufficiently dramatic, and so much more rapid than any obtained by the usual cardiac stimulants that it seemed reasonable to try the effect of similar injections in other cases for the specific purpose of raising the blood-pressure. Cases of spinal anaesthesia with a sudden initial fall of blood-pressure before the operation had begun and before there was any actual blood lost

outside the body were thought to be particularly suitable. It was with regard to the problem of making spinal anæsthesia as safe for the patient as it makes operating easy for the surgeon that this investigation was undertaken, and with special reference to the use of spinal anæsthesia for acute intestinal obstruction. In these cases it is of great value, but it may be dangerous for the patient who is already very ill. The advantages are that it ensures absolute muscular relaxation, which can otherwise only be obtained by deep inhalation anæsthesia of toxic substances undesirable for a toxic patient. It is safer for the patient to be conscious and able to cough and so avoid inhaling regurgitated material. The distension of the intestines is reduced and they are not extruded from the wound with each respiration. Paralysis of the sympathetics by spinal anæsthesia may cause an evacuation of the bowels as soon as the obstruction is removed. This is desirable in preventing further absorption of toxins from the fluid contents of the intestines. Breathing is quiet with spinal anæsthesia, since the intercostal nerves are blocked to a definite level, and there is no straining when the peritoneum is closed. The serious disadvantage in using spinal anæsthesia is the considerable fall in blood-pressure coincident with the onset of anæsthesia. In a case of intestinal obstruction the patient is already in a condition of shock with dehydration and loss of chlorides from the blood by vomiting, and poisoned by the absorption of toxins from the intestinal contents. He is in no condition to stand any further lowering of his blood-pressure.

THE TYPE OF SHOCK IN SPINAL ANÆSTHESIA

In 300 cases operated upon under spinal anæsthesia, in 157 (32·3 per cent) there was an initial fall in blood-pressure before the operation began in spite of some stimulating drug having been injected prophylactically. Even in an averagely healthy patient who, for example, is having an interim appendicectomy performed, there is generally a tendency for the blood-pressure to be lowered for ten to fifteen minutes after the intrathecal injection of the anæsthetic solution. This appears to bear no relation to the drug used nor to its specific gravity, but depends upon the level of anæsthesia and muscular paralysis produced. In the group of 300 cases mentioned above, there was little or no anæsthesia produced in 42 cases (14 per cent). In only 2 of these was there a fall in blood-pressure before the operation began. The sudden hypotension may cause pallor, sweating, nausea, retching or vomiting, with anxiety, restlessness, or delirium. When this condition, described by Quarella¹⁰ as 'the storm', is due to the anæsthesia alone spontaneous recovery may be expected to take place in twenty-five to thirty minutes. For this reason Sebrechts¹¹ states that it is dangerous to begin to operate within thirty-five minutes of the intrathecal injection. Howard Jones¹² explained the collapse by the absorption of the anæsthetic drug into the general circulation, but absorption from the cerebrospinal fluid to the blood is slow. In the cases of failed spinal anæsthesia where, although the usual quantity of anæsthetic has been injected with the usual technique, little or no anæsthesia or paralysis of muscles occurs, there is the same opportunity for absorption from the cerebrospinal fluid, but no fall in blood-pressure is recorded. The possibility of puncturing a vein on performing lumbar puncture, and injecting directly into it, could only occur with faulty technique, by not allowing a flow of clear cerebrospinal fluid before making the injection. This explanation of the collapse is not the most probable one.

The condition of shock coincides with the rapid onset of anæsthesia and flaccid paralysis of more than half of the muscles of the body. The two factors which appear to determine the distribution of the volume of circulating blood at this time are, firstly, the extensive flaccid muscular paralysis, and, secondly, paralysis of the sympathetics. Blood may stagnate in the vessels of the affected skeletal muscles, and in the dilated vessels of the splanchnic area, with exudation of plasma into the tissue spaces. The volume of circulating blood is reduced and is more concentrated. There is a natural tendency to restore the circulating blood-volume by drawing on the fluid reserves of the body and the recovery phase sets in.

In experimental shock Blalock¹³ found that there was accumulation of blood with local loss of plasma in the traumatized areas on comparing the weights of the crushed hind limb of a dog with its normal fellow. He found that trauma never reduced the blood-pressure to a shock level without causing the loss of enough blood and plasma into the traumatized area to account for this fall. This work was confirmed by Holt and Macdonald.¹⁴ Their experimental findings seem to correspond with the apparent distribution of blood at the onset of spinal anæsthesia, although at present absolute proof is lacking. A small practical point supporting this view is mentioned by Hughes,¹⁵ who finds that by bandaging the lower limbs striking falls in blood-pressure are prevented. The part played by the adrenal cortex and medulla in the production of this condition of shock can only be suggested by analogy with the circulatory collapse and hypotension of Addison's disease, which it resembles.

METHODS USED IN THE PREVENTION AND TREATMENT OF THE FALL IN BLOOD-PRESSURE IN SPINAL ANÆSTHESIA

Blood-pressure may be raised by: (1) Increasing the force of the heart's contraction; (2) Increasing the peripheral resistance by vasoconstriction; (3) Increasing the depth of respiration; (4) Increasing the volume of the blood circulating.

Prophylactic injection of a pressor substance is the method commonly used to prevent the initial fall in blood-pressure. Some of the proprietary preparations of novocain contain adrenalin or strychnine, so that it is all given intrathecally. This seems to be of doubtful value because of the slow absorption of drugs from the cerebrospinal fluid into the general circulation. Ephedrine has been given alone or combined with a local anæsthetic solution for injection subcutaneously and intramuscularly at the site of the proposed lumbar puncture. It has been found to have a better stimulating effect on the circulation in a dose of $1\frac{1}{2}$ gr. in preference to two or more smaller doses. It should be given at the same time as the spinal anæsthetic, or before, so that its maximum effect coincides with the onset of anæsthesia. Given later it frequently fails to raise a lowered blood-pressure, and the pressor effect is less with repeated injections. Racedrin (Bayer), racemic ephedrine, appears to be more reliable in its action.

Adrenalin and pituitrin are too powerful and too transient in their action to be of routine use, but may occasionally be of use in a sudden emergency. Coramine (Clayton Aniline Company) has been found to be a powerful respiratory stimulant without any following circulatory depression,¹⁶ but it should be given in full doses of 3 to 4 or 5 c.c. intramuscularly or intravenously where there is severe collapse.

Icoral (Bayer), a solution of two synthetic bases—one like lobeline with a stimulating action on the respiratory centre, the other like ephedrine—stimulates the circulation but is more powerful, and the action takes place in five or ten minutes and lasts longer.

These methods of raising the blood-pressure by means of possibly some slight stimulation of the cardiac muscle, but chiefly by constricting the arterioles and raising the peripheral resistance, may be best employed prophylactically. If there is constriction of the arterioles at the onset of anæsthesia, there is less opportunity for the blood to stagnate in the paralysed lower limbs, but the spinal anæsthetic paralyses the sympathetics, antagonizing the action of the drugs injected unless they are working very strongly first.

An objection to the use of vasoconstrictors for raising the blood-pressure when shock has occurred is that the oxygen supply to the tissues is diminished by virtue of the constriction, and this at a time when more oxygen is required.

Increased depth of respiration raises the blood-pressure firstly by the increased action of the diaphragm. The suction of the thoracic contents aids the return of venous blood to the heart; a greater volume is pumped with each beat and the heart's output is increased. Secondly, there is increased oxygenation of all the tissues, including the heart muscle by the coronary arteries, and the adrenals, as pointed out by Flint¹⁷ in his article on the use of carbon dioxide for this purpose. Some of the drugs discussed previously owe their action on the blood-pressure in part to their action on the respiratory centre in the medulla.

The continuous administration of varying percentages of carbon dioxide and oxygen increases the depth of respiration by direct action on the respiratory centre, and will raise the blood-pressure some 20 to 30 mm. Hg. This will not be sufficient to combat severe shock with very low readings.

Pure oxygen inhalation produces a rise in systolic blood-pressure without increased depth of respiration. Improved oxygenation of all the tissues will account for this.

Nitrous oxide and oxygen anæsthesia has also been found beneficial in raising the blood-pressure, owing to several combined factors. Nitrous oxide itself in small quantities has a stimulating effect; oxygen is then supplied with it and acts in the same way; a re-breathing bag is usually employed for administration, therefore a certain percentage of carbon dioxide is present and exerts its similar effect. Lastly, the fact that the patient is now completely unconscious means that there can be no further psychic shock.

The posture of the patient may be mentioned at this point, as it may affect the respiratory movements. A great deal of attention has been paid in the past to the position of the patient while administering and maintaining spinal anæsthesia. A certain amount of care and common sense is required on account of the relative density of the fluids used and the action of gravity affecting the spread of anæsthetic fluid. After about twenty minutes the anæsthesia appears to become fixed and the patient may be moved into the position most convenient for the surgeon. It is customary to treat shocked patients by blocking the foot of the bed, or by using the Trendelenburg position on the operating-table to encourage the circulation to the brain and vital centres. A tilt of 5° or 10° usually helps the patient, but a steeper incline, although strongly recommended by some authorities, may have the opposite effect to that desired. The action of the diaphragm may be impeded by the weight

of the abdominal contents, and this is particularly true with obese patients whose hearts are loaded with fat. The venous return to the heart is not so good, and sometimes the lower blood-pressure records are met when the patient is steeply tilted.

The question of the volume of fluid in the circulation being maintained at a normal level is of great importance. When the fluid is actually lost from the body by hæmorrhage, it is best restored by transfusion of whole blood. When this cannot be obtained, isotonic saline solution with gum acacia, the colloidal properties⁵ of which prevent the passage of the solution out of the circulation, may be given intravenously instead, but it lacks hæmoglobin and antibodies of value in the recovery phase. These are the measures preferred at the end of a long operation where there has been blood-loss.

In cases of intestinal obstruction fluid and salt are lost from the body by vomiting. There is thus a condition of dehydration with exudation of plasma into the tissue spaces leaving behind a diminished volume of blood in the circulation with a high percentage of hæmoglobin, a raised blood-urea, and lowered chloride content. Before operation the condition is improved and the blood-pressure raised to some degree by the infusion of one or more pints of isotonic saline. Of interest in connection with the rate at which normal saline leaves the circulation is the experimental work of Boycott,¹⁸ and also of Smith and Mendel.¹⁹ They injected Ringer's solution into the jugular vein of rabbits without bleeding them. The dilution of the blood was tested at five-minute intervals and showed that it had returned to the normal volume in twenty-five minutes; 67 per cent of the fluid escaped within five minutes, and there was no œdema or pleural effusion. Engels²⁰ found that the muscles, which are approximately 40 per cent of the total body weight, contained 60 per cent of the fluid after intravenous injection. He was using dogs which had been deprived of water and food for four days. Whether the solution is used with gum acacia or not, when the spinal anæsthetic is administered the blood-pressure is likely to become lowered as it takes effect, but probably not to the same extent as without a preliminary intravenous infusion. Other factors play their part and must be considered—namely, the age and nutrition of the patient, the length of time the obstruction has been present, and its position.

For the reason that under spinal anæsthesia there appears to be a temporary loss of fluid from the circulation into the paralysed muscles and tissue spaces and not an actual loss of fluid from the body, it seems reasonable to use a small quantity of strongly hypertonic sodium chloride solution which as an electrolyte exerts a high osmotic pressure and rapidly restores fluid to the circulation and tides over a period of crisis. No extra fluid volume is used which can embarrass the heart's action, and no vasoconstriction occurs to diminish the oxygen supply to the tissues.

On injecting 40 c.c. of 30 per cent sodium chloride solution into a vein in a patient suffering from the initial collapse following spinal anæsthesia, there is usually a dramatic response in five minutes which is more rapid than any found with the circulatory stimulant drugs mentioned above. The patient, instead of being restless, anxious, sweating, retching, and cyanosed, becomes pink, takes a deep breath, and if conscious will be comfortable and tell you he feels better. The pulse, from being impalpable or very weak, increases in volume and becomes slower in rate. This can be checked by blood-pressure readings taken at two-and-a-half-minute intervals after the injection. The systolic pressure rises rapidly and there is a greater

pulse-pressure. The clinical picture resembles very closely the response of a patient with Addison's disease in crisis to treatment with intravenous sodium chloride and cortical adrenal extract, the difference being that in the cases under consideration the adrenal glands are presumably not permanently diseased, but may be temporarily disturbed.

SOME PHYSIOLOGICAL EFFECTS OF HYPERTONIC SODIUM CHLORIDE

When hypertonic sodium chloride solution is injected into a vein it exerts a high osmotic pressure. Water is drawn from the tissues into the circulation during the period when the plasma is hypertonic.²¹ There is a transitory increase in the plasma volume and dilution of the plasma proteins. The colloidal osmotic pressure is reduced, diuresis results, and the blood-volume may then fall below normal by the water lost in the urine. The diuresis itself may be of use in helping the body to get rid of acid products and toxic substances produced while there is diminished activity of all tissues during the period of shock when there is a reduced oxygen supply and consequent accumulation of waste material.

Hypothetically sodium chloride may combine with toxin to form a non-toxic product, with a resulting alkalosis. Richet, quoted by Barber and Oriel,²² has shown that experimental anaphylactic shock could be prevented by the previous injection of sodium chloride. It has, on the other hand, been shown experimentally that the flow of urine which follows the intravenous injection of hypertonic saline is checked if a condition of acidosis is produced. The uræmia syndrome²³ occurs through a disturbance in the balance of acid base and mineral salt. No cases of uræmia were noted in the present series of cases, but blood-urea estimations were not made. Those deaths occurring soon after operation were in cases which were almost hopeless operative risks and could not be attributed to hypertonic sodium chloride.

There is a change in colour on increasing the sodium chloride²¹ in plasma. Varying carbon dioxide tension in the blood alters the distribution of chlorine as well as HCO_3 between cells and plasma. If the carbon dioxide tension is raised, more H_2CO_3 is formed, combining with part of the base previously bound by hæmoglobin, forming potassium bicarbonate in place of potassium hæmoglobinate. The electrolytic action of NaCl may cause a reversal of the reduced hæmoglobin to normal oxyhæmoglobin. Clinically the rapid change from pallor and cyanosis to a pink colour is seen immediately in the face and hands when extra sodium chloride has been given. The explanation may not be entirely chemical, but in part due to the raised blood-pressure causing better oxygenation of the blood in the lungs.

A complaint of thirst is made by many patients soon after the injection of hypertonic saline. The dryness of the throat and defective secretion of saliva may be explained by the osmotic mechanism of the secretory cells by which water is passed from the blood to the ducts functioning under greater difficulties because more work is required to remove water from a concentrated solution than from a dilute.

Sudden severe headache may occur immediately after the intravenous injection has been made, and must be attributed not so much to the sudden lowering of intracranial tension as to the sudden increased blood-supply to the brain before the change in flow of cerebrospinal fluid occurs.²⁴ It is a very temporary headache and usually has passed off within ten minutes and does not recur.

Increased peristalsis and evacuation of the bowels occurred in a relatively low percentage of cases, but the small intestine may empty itself into the large bowel without complete evacuation. Necrosis of the skin occurred in 3 cases where a small quantity of the hypertonic saline was injected subcutaneously by an error of technique. The skin becomes dehydrated and changes colour from normal to dead white to brown within a few minutes. Later a slough separates. Thrombosis of a vein occurred in two cases where small veins were used and the injection was made with difficulty and slowness. No thrombosis occurred where large-sized veins were used such as those at the bend of the elbow.

CASE RECORDS

Of 53 cases where the blood-pressure was recorded after the intravenous injection of hypertonic sodium chloride solution, 39 were operated upon under

Table I.—NINETEEN CASES TREATED BY INTRAVENOUS INJECTION OF 30 PER CENT SODIUM CHLORIDE FOR SHOCK OCCURRING DURING OPERATION FOR INTESTINAL OBSTRUCTION PERFORMED UNDER SPINAL ANÆSTHESIA

CASE NO	SEX AND AGE	INITIAL BLOOD-PRESSURE	SYSTOLIC BLOOD-PRESSURE BEFORE NaCl	C C OF 30 PER CENT NaCl	SYSTOLIC PRESSURE			REMARKS
					5 min after	10 min after	30 min after	
2	F. 60	140/100	70	40	120	130	140	Gum saline followed on Coramine and pituitrin no action in 25 minutes
6*	M. 75	110/60	zero	50	100	—	140	
7*	M. 59	140/100	60	40	105	110	120	Blood-sodium during shock = 311
9*	M. 52	170/100	85	40	160	180	140	Blood-chlorine during shock = 347
10	F. 46	130/80	80	40	100	100	80	Blood-sodium during shock = 302
12	F. 22	115/90	50	30	70	70	—	Blood-chlorine during shock = 385
14*	M. 73	105/70	105	30	150	125	140	Blood-sodium during shock = 344
								Evipan-sodium anæsthesia in addition
								Injection made before spinal anæsthesia
15*	F. 54	160/100	50	80	60	60	—	Responded to icoral rather than to NaCl
			60	15% 40	80	70	—	
21*	F. 30	115/80	60	30% 30	100	100	—	Shock developed towards end of operation
22	F. 76	140/80	zero	40	80	50	—	Died 2 hours later
27	M. 73	145/80	60	50	80	80	—	Died 6 hours later
30*	M. 32	140/100	70	40	100	120	80	Followed by gum saline
32	F. 83	140/90	60	40	120	200	150	Combined action with icoral previously injected but which had shown no effect in 5 minutes
33	M. 53	120/65	50	40	—	100	120	Previous injection of 40 c c 50 per cent glucose
36	M. 21	120/70	80	40	110	115	100	
37*	M. 54	145/80	70	40	110	125	—	
38*	F. 52	160/100	zero	40	100	110	100	Previous injection of 80 c c 50 per cent glucose
39*	F. 49	120/70	95	40	150	175	—	
48	F. 69	130/70	60	40	100	110	100	

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity

spinal anæsthesia, and 19 of these were for intestinal obstruction. There were 6 operations for cerebral decompression under nitrous oxide and oxygen anæsthesia, 4 other cases under gas and oxygen, and 2 under local anæsthesia. There were 2 post-operative cases. The records will be considered in these groups for better comparison.

Among the group of operations for intestinal obstruction, in 3 cases (*Cases 7, 9, and 10*) blood was taken for the estimation of sodium and chlorine at the time when the blood-pressure was low. The figures obtained were 311, 302, 344 mgrm. sodium per 100 c.c. serum respectively. It was hoped to have continued these investigations with estimations of hæmoglobin, blood-urea, and blood-sugar, but practical difficulties make it impossible at present.

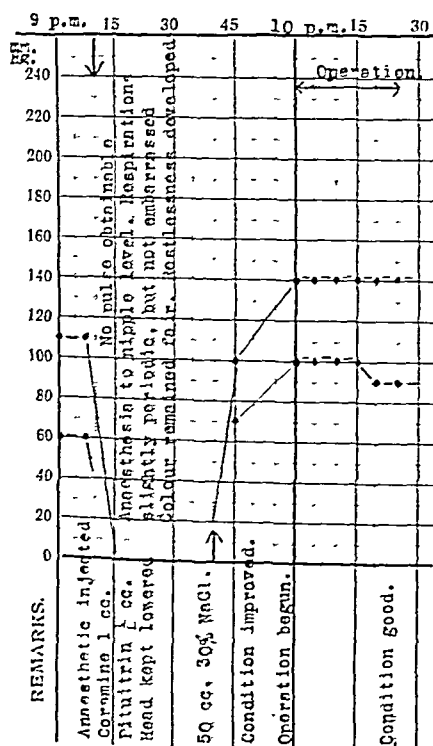


FIG. 81.—Case 6. Male, aged 75.
Gall-stone ileus.

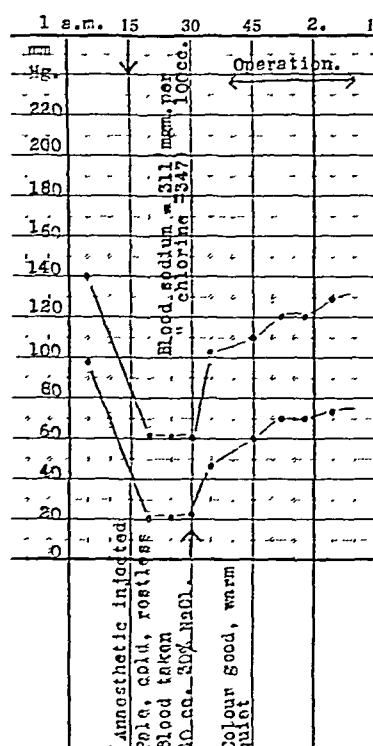


FIG. 82.—Case 7. Male, aged 59.
Adhesions.

Case 6 is of interest in that 2 c.c. of racedrin given with the spinal anæsthetic and 1 c.c. of coramine and $\frac{1}{2}$ c.c. of pituitrin had no effect even after twenty minutes, but 50 c.c. of 30 per cent sodium chloride raised the systolic pressure from zero to 100 mm. Hg in five minutes, with rapid recovery of the patient. The anæsthesia in this case was to a high level; presumably the hypertonic saline mobilized fluid from the paralysed muscles.

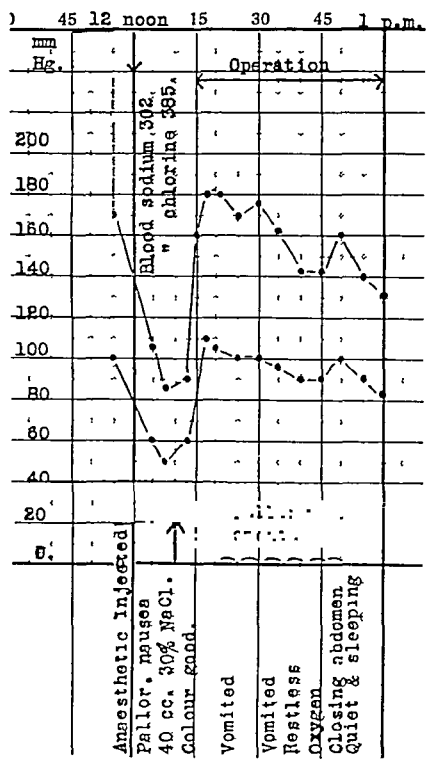


FIG. 83.—Case 9. Male, aged 52. Adhesions.

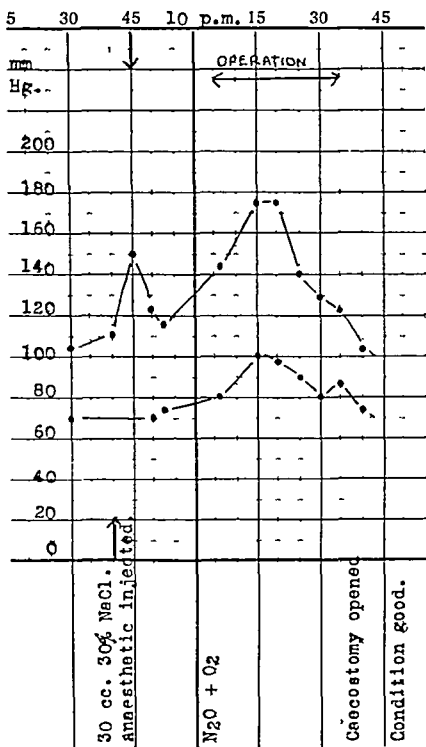


FIG. 84.—Case 14. Male, aged 73. Caecostomy for carcinoma of colon.

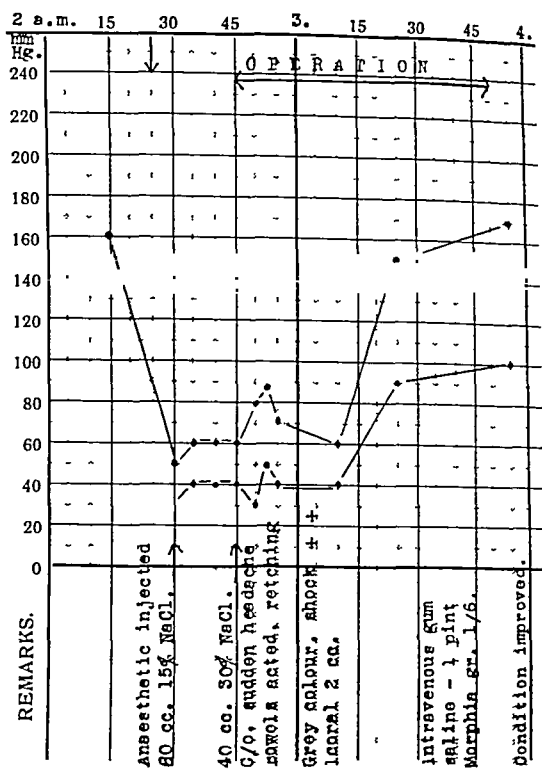


FIG. 85.—Case 15. Female, aged 54. Entero-anastomosis.

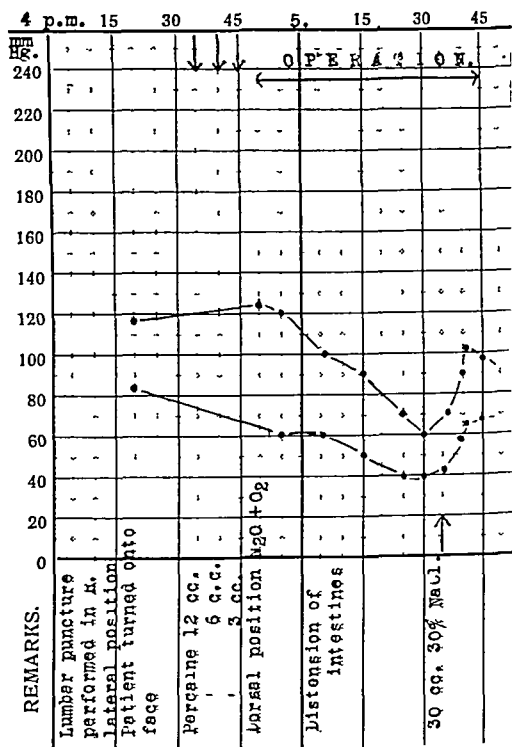
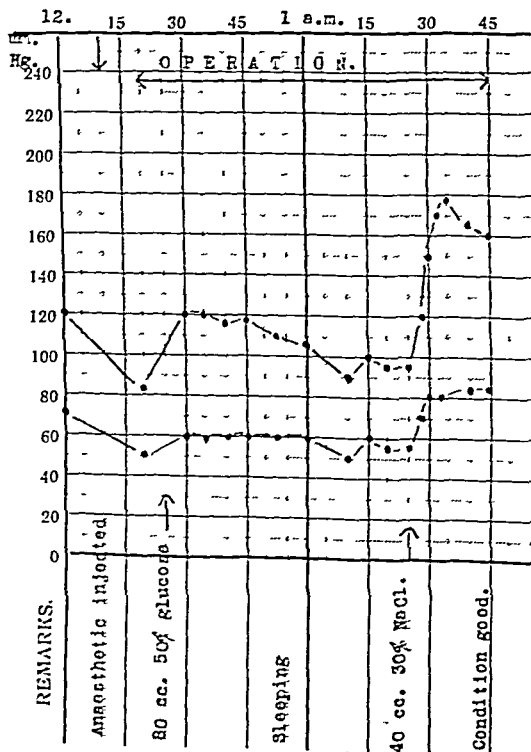
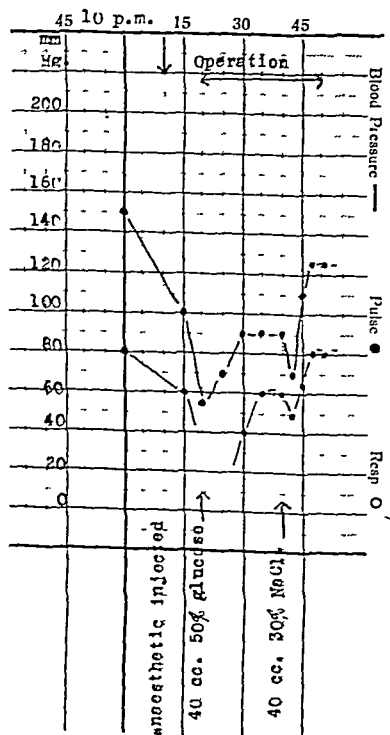
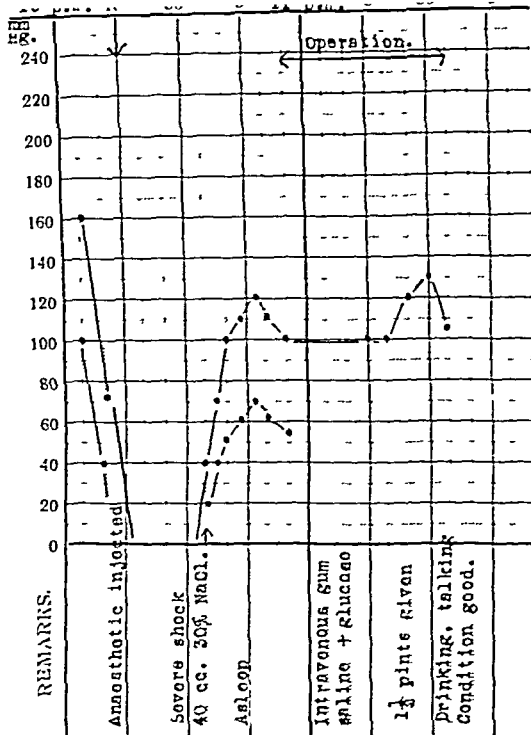
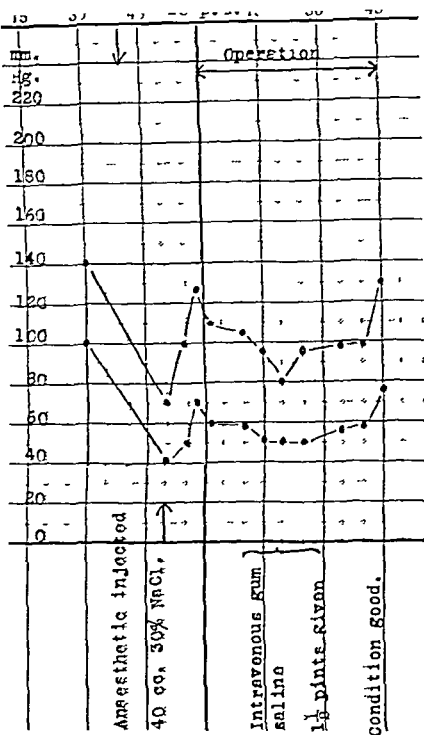


FIG. 86.—Case 21. Female, aged 30. Caecostomy for carcinoma of colon.



Case 14 shows the effect of gas and oxygen in raising the blood-pressure.

Case 15 shows that 80 c.c. of 15 per cent sodium chloride had very little effect, 40 c.c. of 30 per cent raised the systolic pressure by 30 mm. Hg by its greater osmotic pressure, but in this case a cardiovascular stimulant was needed and 2 c.c. of icoral restored the blood-pressure to its normal level.

Cases 37 and 39 show the less powerful effect of a non-electrolytic solution even when hypertonic; 40 c.c. and 80 c.c. of 50 per cent glucose were injected intravenously, but neither raised the blood-pressure to the same level as 40 c.c. of 30 per cent sodium chloride which was given later.

Table II.—TWENTY CASES OF SHOCK OCCURRING DURING SPINAL ANÆSTHESIA TREATED BY INTRAVENOUS INJECTION OF 30 PER CENT SODIUM CHLORIDE SOLUTION

CASE NO	SEX AND AGE	INITIAL BLOOD-PRESSURE	SYSTOLIC BLOOD-PRESSURE BEFORE NaCl	C C OF 30 PER CENT NaCl	SYSTOLIC PRESSURE			REMARKS
					5 min after	10 min after	30 min after	
3	M. 66	120/60	80	20	105	105	—	Perforated gangrenous appendix
4	M. 41	140/80	50	20	85	85	—	Pyelolithotomy
5*	F. 54	140/80	100	30	180	175	140	Diverticulitis
11*	F. 5	130/80	50	15	95	120	95	Case of adrenal hyperplasia
			40	20	80	110	110	
16	F. 76	130/70	zero	50	80	120	—	Diathermy excision of vesical growth
18	F. 48	120/80	70	40	95	110	95	Large ovarian cyst
20	F. 59	100/80	45	20	90	85	70	Mikulicz operation, carcinoma colon
23	M. 35	130/80	85	40	100	130	—	Gangrenous appendix plus bronchitis
24	M. 24	150/90	90	40	110	130	145	Gangrenous appendix
25	M. 51	120/90	60	40	80	120	—	Appendicostomy for colitis
28	M. 53	120/70	70	40	110	150	140	Mikulicz operation, carcinoma colon
29	M. 71	100/60	90	40	140	125	—	Amputation leg—diabetic gangrene
42	F. 69	155/90	80	40	90	150	125	Colectomy for carcinoma
43	M. 34	120/80	95	40	135	145	130	Colectomy for carcinoma
44	M. 51	160/80	60	40	100	120	—	Perforated diverticulitis
45	F. 54	120/80	50	40	100	110	80	Gastro-enterostomy—carcinoma pancreas
46	M. 58	125/80	90	40	100	120	115	Excision of rectum
51*	M. 43	110/60	80	40	138	118	105	Partial gastrectomy
			80	40	120	120		
52	M. 39	160/110	75	40	125	125	130	Large adherent hydronephrosis
53*	M. 56	160/100	70	40	120	160	140	Cholecystectomy—jaundice

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity.

Cases 11 and 51 both show that a second injection of hypertonic saline will have a similar effect on the blood-pressure. In each case after the first administration the blood-pressure remained steady until a further noxious stimulation caused further shock; by altering the osmotic pressure apparently fluid is restored from the tissue spaces into the circulation and after some sort of adjustment tends to return to normal and not to the shock condition.

The little girl aged 5 (Case 11) was of peculiar interest. She was explored for a possible adrenal tumour as she showed signs of virilism. Actually hyperplasia was present and one gland was removed. Each time the blood-pressure fell

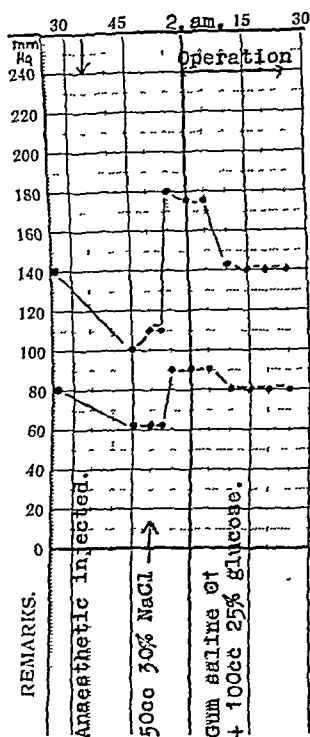


FIG. 91.—Case 5. Female, aged 54. Diverticulitis.

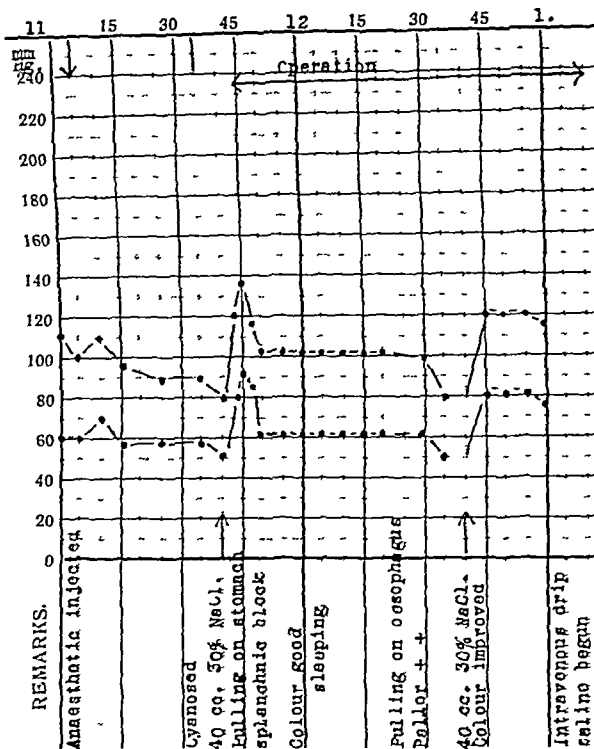


FIG. 93.—Case 51. Male, aged 43. Partial gastrectomy.

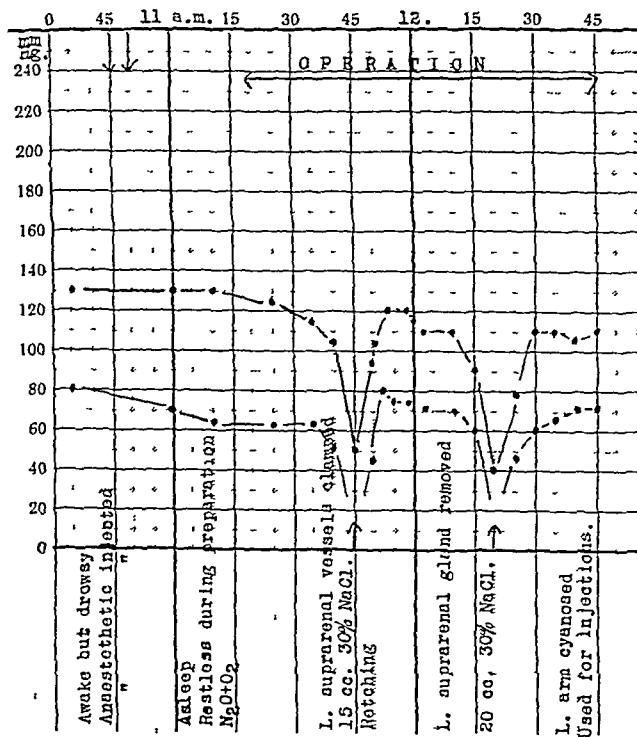


FIG. 92.—Case 11. Female, aged 5. Exploration—hyperplasia of adrenals

suddenly there had been interference with the adrenals. There was gross paralytic distension of the stomach and intestines and neither the spinal anæsthetic nor

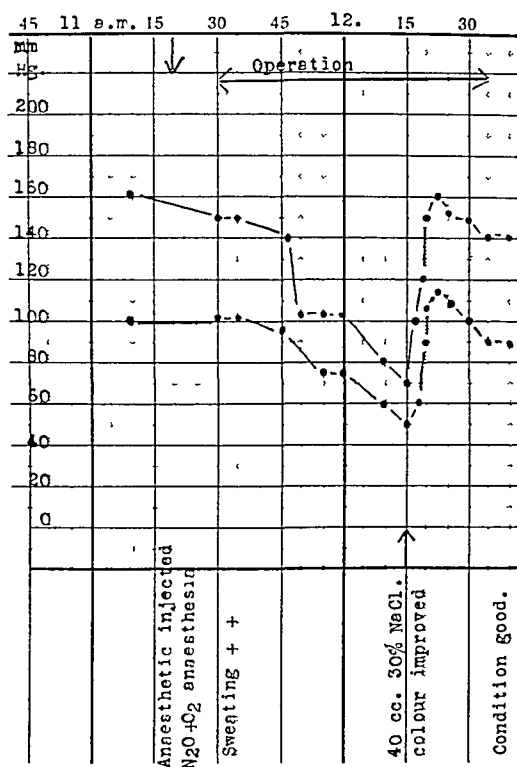


FIG. 94—Case 53. Male, aged 56. Cholecystectomy. Stone in common duct. Jaundice.

Table III.—THE EFFECT OF 30 PER CENT SODIUM CHLORIDE INJECTED INTRAVENOUSLY TOWARDS THE END OF OPERATIONS FOR CEREBRAL DECOMPRESSION, WHERE THE INTRACRANIAL TENSION WAS RAISED

CASE No.	SEX AND AGE	DURATION OF OPERATION BEFORE INJECTION	BLOOD-LOSS	BLOOD-PRESSURE BEFORE INJECTION	C C. 30 PFR CENT NA CL	BLOOD-PRESSURE			REMARKS
						5 min after	10 min after	30 min. after	
I	M. 68	2 25	Slight	130/90	30	160/100	140/90	140/100	Left frontal tumour
34	F. 41	2 40	++	80/55	50	138/80	140/90	130/90	VIIIth nerve tumour
40	M. 29	3 10	+	85/40	50	140/70	140/70	100/60	Traumatic orbital meningocele
47	M. 52	1 35	—	80/55	40	118/80	118/80	—	Right frontal 2ndy. tumour with arterial hypertension
49	M. 52	1 10	—	85/?	40	105/80	125/85	140/95	Right frontal meningioma with arterial hypertension
50	F. 44	2 0	—	160/110	40	185/110	190/118	—	Right temporal lobe tumour

hypertonic saline had any effect in lessening it. Her pulse-rate was rapid, nearly 160, most of the time she appeared to be under predominant sympathetic stimulation.

Of interest in the group of cases in *Table III* is the fact that the same rise in blood-pressure occurs on injecting hypertonic saline even after a considerable loss of blood. In *Case 34* a blood transfusion had just been begun when the injection was made, and in *Case 40* gum saline was being given. In neither case had the infusions raised the low blood-pressure. In *Case 50*, where the blood-pressure was already high, a further increase occurred. There appeared to be no diminution in the intracranial tension.

Table IV.—HYPERTONIC SALINE GIVEN IN CASES OF SHOCK OCCURRING DURING OPERATIONS PERFORMED UNDER : (1) NITROUS OXIDE AND OXYGEN ANÆSTHESIA ; (2) LOCAL ANÆSTHESIA

CASE No.	SEX AND AGE	DURATION OF OPERATION BEFORE INJECTION	BLOOD-LOSS	BLOOD-PRESSURE BEFORE INJECTION	C.C., 30 PER CENT NaCl	BLOOD-PRESSURE			REMARKS
						5 min. after	10 min. after	30 min. after	
1. Nitrous Oxide and Oxygen Anæsthesia.—									
17	M. 31	Hr. Min 1 50	+	90/50	50	100/50	118/60	100/50	Forequarter amputation for sarcoma left axilla
19	M. 61	1 0	—	60/45	40	100/60	130/80	—	Jejunal ulcer
26*	F. 48	—	—	40/?	40	140/100	180/120	140/90	Acute pancreatitis, shock + +, pre-anæsthetic injection of NaCl
31	F. 62	1 0	—	80/50	40	120/60	140/90	110/70	Ileostomy
2. Local Anæsthesia.—									
8	M. 72	—	—	110/80	20	140/90	138/85	—	Cæcostomy, local anæsthesia, arteriosclerosis + +
13	M. 75	—	—	80/70	40	120/70	130/70	50/?	Extreme collapse, strangulated umbilical hernia, drained

* Detailed chart of systolic and diastolic blood-pressure given. The pulse-rate record has been omitted for the sake of clarity.

Case 26, a woman of 46 suffering from acute pancreatitis, was severely shocked and restless. In spite of morphia and a pint of gum saline solution given intravenously before there was any interference, her systolic blood-pressure was only 40 mm. Hg. There was an immediate response to 40 c.c. of 30 per cent sodium chloride which enabled laparotomy to be performed.

Two post-operative cases were given hypertonic saline intravenously. *Case 35*, a woman of 40, was shocked following vaginal hysterectomy by a reactionary intraperitoneal hæmorrhage. Her pulse could not be felt and she responded to no treatment for three or four hours, when recovery gradually came spontaneously. The only time her pulse was felt during this period was immediately after injecting 40 c.c. of 30 per cent sodium chloride. *Case 41* was a man of 37 with paralytic

ileus due to general peritonitis from appendicitis; 80 c.c. of 15 per cent sodium chloride were given intravenously to stimulate peristalsis. It failed to do this, but the blood-pressure rose from 130/80 to 150/90 in five minutes.

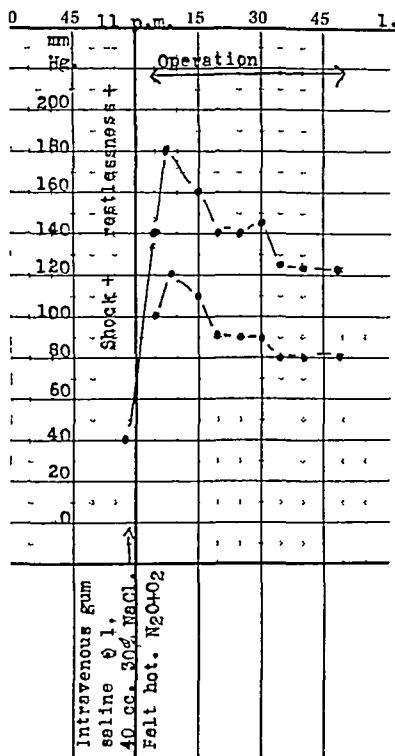


FIG. 95—Case 26 Female, aged 48. Acute pancreatitis.

SUMMARY

1. The therapeutic uses of hypertonic sodium chloride solution are mentioned.
2. The advantages and dangers of spinal anaesthesia with special reference to its use in cases of intestinal obstruction are discussed.
3. The type of shock present in spinal anaesthesia is considered to be similar to the experimental traumatic shock studied in animals where the blood stagnates in the injured limbs, and in spinal anaesthesia in the anaesthetized and paralysed regions. Plasma escapes into the interstitial spaces, and the circulatory collapse may be comparable with that seen in Addison's disease.
4. The administration of vasoconstrictor drugs to raise the blood-pressure when shock has developed may be ineffectual and undesirable on account of the further reduction of the oxygen supply to the tissues and vital organs.
5. Extra fluid introduced into the circulation is good pre-operative treatment in cases of intestinal obstruction where there is dehydration, but may not prevent the fall in blood-pressure caused by the onset of spinal anaesthesia.
6. In order to mobilize fluid from the tissue spaces, hypertonic saline solution (usually 40 c.c. of 30 per cent sodium chloride) has been injected intravenously.

The circulation was stimulated immediately, with improvement in the patient's condition. In five minutes a considerable increase in blood-pressure could be recorded. The rise was towards the level normal for the individual. Hypertension was not found, as may occur when using the more potent vasoconstrictor drugs. After twenty to thirty minutes when the body fluids had become re-adjusted the blood-pressure readings were on a lower level, but no longer at a shock level unless this was freshly induced by the operative procedure.

The intravenous injection of a small quantity of strongly hypertonic sodium chloride solution appears to be a reasonable method of treating the low blood-pressure occurring at the onset of spinal anæsthesia, and has been found of practical benefit to the patient in tiding over a period of crisis.

My thanks are due to the surgeons of the Leicester Royal Infirmary for their permission to inject hypertonic saline in these cases.

REFERENCES

- ¹ WEED, "Experimental Alterations in Brain Bulk", *Amer. Jour. Physiol.*, 1919, xlviii, 512, 531.
- ² OCHSNER, "Motility of the Intestine", *Arch. of Surg.*, 1933, xxvii, 742.
- ³ ROGERS, *Tropical Medicine*, 1935, 290. London: Churchill.
- ⁴ GRAHAM, *St. Bart's Hosp. Rep.*, 1929, lxii, 55.
- ⁵ BAYLISS, *Intravenous Injection of Wound Shock*, 1918. London: Longmans, Green & Co.
- ⁶ SWINGLE and PFIFFNER, *Amer. Jour. Physiol.*, 1933, 108-144.
- ⁷ LOEB, ATCHLEY and STAHL, "Role of Sodium in Adrenal Insufficiency", *Jour. Amer. Med. Assoc.*, 1935, civ, 2149, 2177.
- ⁸ MACFEE and BALDRIDGE, "Infusion Treatment of Shock", *Ann. of Surg.*, 1934, c, Aug., 266.
- ⁹ CUSHING and FOLEY, *Proc. Soc. Biol. and Med.*, 1920, xvii, 217.
- ¹⁰ QUARELLA, "Spinal Anæsthesia with Percaine", *Minerva Med.*, 1930, Sept.
- ¹¹ SEBRECHTS, Technique described by J. W. West, *Jour. R.A.M.C.*, 1933, July.
- ¹² JONES, HOWARD, *Brit. Jour. Anæsth.*, 1930, vii, April and July.
- ¹³ BLALOCK, "Cause of Low Blood-Pressure Produced by Muscle Injury", *Arch. of Surg.*, 1930, xx, 959.
- ¹⁴ HOLT and MACDONALD, "Observations on Experimental Shock", *Brit. Med. Jour.*, 1934, i, 1069.
- ¹⁵ HUGHES, *Ibid.*, 1935, 2, 3.
- ¹⁶ MONCRIEFF, "Respiratory Failure", *Lancet*, 1935, ccxxviii, 664.
- ¹⁷ FLINT, "Experiences with Spinal Anæsthesia", *Brit. Med. Jour.*, 1935, i, 197.
- ¹⁸ BOYCOTT, "Regulation of Blood Volume in Normal and Nephritic Animals", *Jour. Pathol. and Bacteriol.*, 1913, xviii, 11.
- ¹⁹ SMITH and MENDEL, "Adjustment of Blood Volume after Injection of Isotonic Solutions", *Amer. Jour. Pathol.*, 1920, liii.
- ²⁰ ENGELS, *Arch. f. exper. Pathol. u. Pharmacol.*, 1904, li, 346.
- ²¹ PETERS and VAN SLYKE, *Quantitative Clinical Chemistry Interpretations*, 1931, i, 665, 1034, 1021. Baltimore: Williams and Wilkins.
- ²² BARBER and ORIEL, "Allergy", *Lancet*, 1928, ccxv, 1009.
- ²³ ANDREWS, "Experimental Uræmia", *Arch. of Internal Med.*, 1927, 548.
- ²⁴ WEED, *Physiol. Rev.*, 1922, ii, 171.

SOME OBSERVATIONS ON THE SURGERY OF TRIGEMINAL NEURALGIA

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THE first intracranial operation for the relief of idiopathic trigeminal neuralgia was performed in this country by Victor Horsley¹ in 1891. Horsley approached the nerve by the intradural parietal route and resected a portion of the Gasserian ganglion. The patient died, though post-mortem examination showed that there was no gross brain-lesion.

Between 1891 and 1901 there were sporadic reports of cases operated on by this method; Tiffany,² for example, fully reports a successful case.

In 1901 Frazier³ introduced the extradural method of approach, and he replaced gasserianectomy by partial resection of the sensory root of the nerve. This gradually became the standard method and is the basis of the practice of most neurological

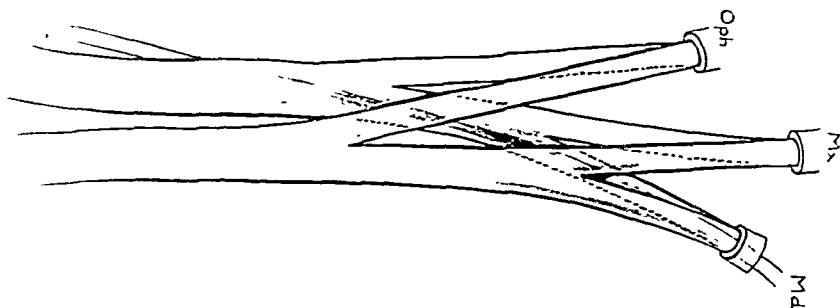


FIG. 96.—To illustrate Dandy's postulate of the disposition of the fibres of the sensory root of the trigeminal nerve. Oph., Ophthalmic division; Mx., Maxillary division; Md., Mandibular division.

surgeons in this country. In 1925, however, Dandy⁴ introduced the method of intradural approach to the nerve by the posterior fossa and, with the new method, he offered several criticisms of the old. He held that the Frazier method must cause anæsthesia as well as analgesia, and claimed that by section of the sensory root close to the pons instead of (as previously) close to the ganglion, he produced analgesia without anæsthesia. In explanation of this result Dandy postulated (though he did not pretend to have proved) a possible re-arrangement and segregation of the fibres in their passage from the ganglion to the pons (*Fig. 96*). Thus the pain-fibres of each division of the nerve separate off from the touch-fibres soon after leaving the ganglion, and come to lie in the posterior (or lateral) half of the nerve; while the touch-fibres similarly collected together occupy the anterior (or

medial) half of the nerve as they approach the pons. This hypothesis would readily explain the difference between the results of the two methods. By dividing about the outer one-third of the nerve close to the ganglion, one would produce analgesia and anæsthesia in the mandibular division. By dividing about the outer one-half of the nerve close to the pons, one would produce general analgesia of all three divisions, but no anæsthesia.

Before reporting the investigations which form the subject of this paper, reference must be made to our previous knowledge of the anatomy of the Vth nerve and its nuclei. With regard to the nuclei it is certain that the principal nucleus is a relay station on the path of epicritic or discriminative fibres, and that the descending or spinal root, relayed in the spinal nucleus, carries pain and thermal impulses. The best evidence for this is the dissociation of symptoms into loss of touch-sensation and loss of pain-sensation seen as the result of lesions involving only one of these two nuclei. Stopford⁵ summarizes the evidence and illustrates the paths of the fibres in his book, and in papers on the function of the spinal nucleus and on the posterior inferior cerebellar arteries. Many other writers—for example, Gillis,⁶ W. Harris,⁷ and M. Gerard,⁸ report individual cases of lesions of the spinal nucleus producing dissociation of sensation (the usual lesion is thrombosis of the posterior inferior cerebellar artery); Stopford⁹ has collected these and added them to his own; and he concludes that the evidence is ample to prove the protopathic function of the spinal fibres and nucleus.

There is no uniformity of opinion on just what the fibres of the sensory root of the Vth nerve are like, or on the details of their fate before or after their entry into the pons. In text-books¹⁰ a general description is given of the fibres of the sensory root as 'dividing into two branches, of which the upper go to the principal nucleus while the lower form the descending or spinal root.' This would imply that protopathic and discriminative impulses are carried by one fibre, and that the sensations only take separate paths when they reach collaterals of the fibre; this conception presents great difficulties. Fortunately Gudden¹¹ describes undivided fibres to a considerable number amongst the divided fibres (about the histological existence of which there is no doubt). So we may suppose on the one hand that some fibres do actually go to both nuclei, and that the difference lies perhaps in threshold of response to stimulation; but that others go only to one nucleus or the other.

Mention should be made of Ranson's argument¹² on physiological grounds, that pain-fibres should be unmyelinated. This may be dismissed for our purposes, since (as shown, for example, by S. L. Koch¹³) the number of non-medullated fibres in the human Vth nerve is very small.

We may take it, then, that while the anatomical observations are not very precise or convincing, the fibres within the pons go to one or other of the two nuclei under discussion.

Kappers,¹⁴ in the course of his most illuminating work on the comparative anatomy of the nervous system of Vertebrata, works out the functions of the different central parts of the Vth nerve. Thus the spinal root and nucleus are the oldest part of the nerve phylogenetically, and in animals below the Reptilia subserve the reflexes of the skin of the head. Only in certain reptiles does the anterior end of the spinal nucleus enlarge and begin to become an entity (principal nucleus), and thus the newer epicritic or discriminatory paths begin. So we may picture the principal nucleus and trigeminal fillet as a contribution to the development of the

neopallial system and the spinal nucleus as the original archepallial non-discriminative system.

For these reasons we may take as a working basis the conception that *somewhere between the Gasserian ganglion and the actual nuclei* the sensory fibres of the Vth nerve undergo a rearrangement and a segregation into discriminatory touch-fibres (principal nucleus) and protopathic fibres (spinal nucleus).

Dandy's hypothesis, then, is only a slight addition to the known facts; it is certain that functional segregation takes place, and Dandy suggests that it takes place in the nerve outside the pons.

There is some clinical evidence in support of this view. Careful examination of a large number of cases of sensory disturbances due to mechanical involvement of the Vth nerve by tumours of the posterior fossa shows that pain sense is lost and tactile sense unimpaired, or relatively unimpaired. A typical case of involvement of the Vth nerve by an acoustic tumour shows a sense of numbness, and definite loss of sensibility to pain; but a pinprick is quite well orientated, but with sensation of touch only. Moderate degrees of heat and cold give variable results, but touch to cotton-wool is definitely appreciated and orientated. Further, the loss of pain sense follows the order one would expect according to Stopford's investigations¹⁵—namely, that the supra-orbital region is first affected, then the cheek, and lastly the mandibular region—corresponding to the known order of fibres from behind forwards. In one case actually a decompression operation (the tumour being deemed irremovable) led to the disappearance of the symptoms in the reverse order—namely, mandibular, buccal, and, lastly, forehead.

These considerations justify the expectation that a section of the outer part of the nerve close to the pons would destroy protopathic fibres, but a similar section close to the ganglion would destroy a topographic portion of the nerve—i.e., some protopathic and some discriminatory fibres arising in a particular region of the face. If these two operations are done experimentally, the first (near the pons) should result in complete degeneration of the spinal root of the nerve; the second (near the ganglion) should result in partial degeneration of the spinal root and partial degeneration of the fibres around the principal nucleus.

Accordingly two series of experiments have been carried out—partial section of the sensory root of the trigeminal nerve by the parietal route, the nerve being cut close to the Gasserian ganglion; and partial section by the posterior-fossa route, the nerve being cut close to the pons.

TECHNIQUE AND RESULTS

Well-grown Macacus monkeys were anaesthetized by induction with C.E. mixture, followed by intraperitoneal dial (1½ c.c.) or nembutal (4 gr.). (Nembutal acts so quickly that the general anaesthetic may be stopped immediately after the injection.)

Method 1.—Frazier's transparietal extradural, with distal section of the nerve (outer one-third of fibres). Four successful operations. Two killed on eleventh and thirteenth day respectively; medulla, pons, and lower midbrain prepared by Marchi's method. *In one case no degeneration at all, in the other so few and scattered fibres as to be of doubtful significance.* The other two were killed on the twentieth and twenty-eighth day respectively and the brain-stem prepared by Weigert Pal. *Again no degeneration found sufficient to be assessed.*

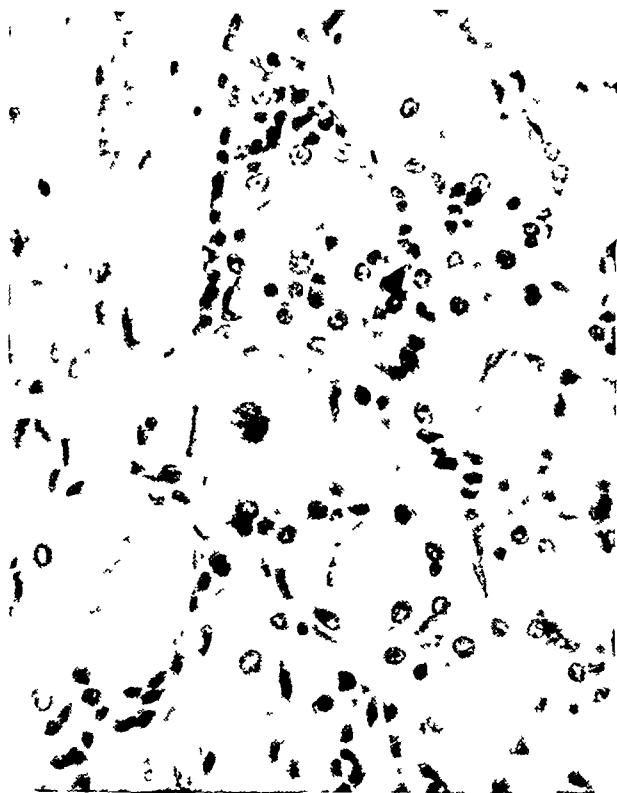


FIG 97—Sensory cells in sensory root of trigeminal nerve near pons Macacus H E (/ 720)

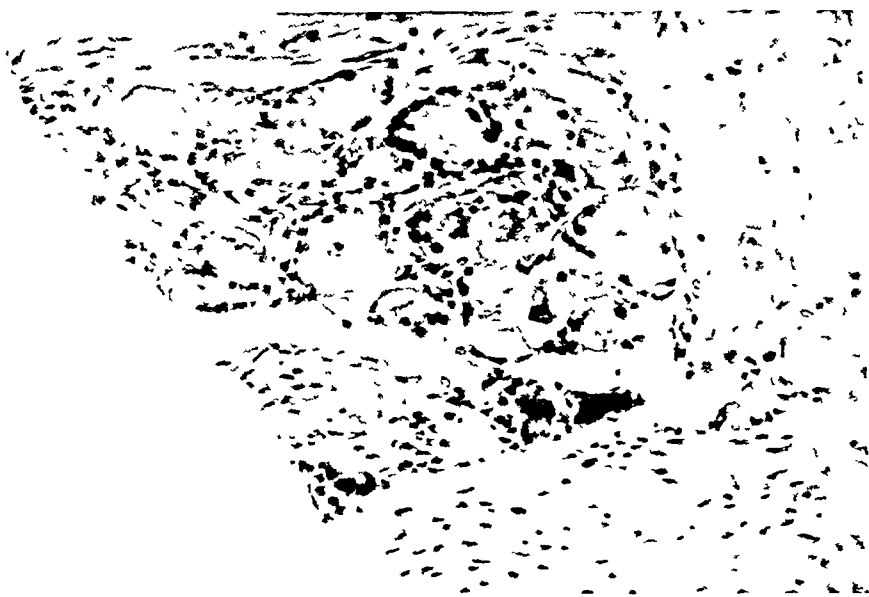


FIG 98—Sensory cells in sensory root of trigeminal nerve near pons Human H E. (\times 360)

These unexpected results were so disconcerting as to make one feel it was necessary to revise one's technique. The explanation, however, came to light during the examination of the stumps of the nerve, which had been preserved. It was found (*Fig. 97*) that the sensory root contained considerable numbers of large sensory nerve-cells at various points of its course, quite separate from the Gasserian ganglion; generally these were in the central half of the root, often quite close to the pons. In the distal operation, therefore, the reason why little or no central degeneration was found, was that the fibres were cut peripherally to their cells of origin. Examination of the Vth nerve in man showed similar groups of centrally-placed cells* (*Fig. 98*). These cells must be regarded as of great significance. The conception that section of the sensory root of the nerve proximal to the ganglion ought permanently to throw it out of action is based on the view that the nerve fibres proximal to the ganglion are a part of the central nervous system and therefore do not regenerate. Actually a proportion of the fibres are cut distal to their cells and might regenerate peripherally. Such regeneration might be the cause of recurrence of pain. It must be added that recurrences nowadays are few, and only after long intervals; but we have, in the presence of these cells, an indication that recurrences are possible when the peripheral operation has been performed.

Method 2.—Dandy's intradural operation via the posterior fossa, with section of the outer half of the nerve close to the pons.

Three successful operations, but only one satisfactory histological result—namely, degeneration of approximately the whole of the spinal root of the nerve with no other degenerations (*Figs. 99, 100*). The reason why the other cases did not succeed histologically is that various other patches of degeneration were found, quite remote from the Vth-nerve tracts. This is due to damage by torsion of the brain-stem during the displacement of the cerebellum; the one successful case was a lucky one in which this had not occurred, but the result is so clear-cut that the writer feels that even one such case is convincing.

Anatomical and histological evidence has been sought for—that is, demonstrations that the fibres do cross as the experimental work indicates. In this connection Frazier states definitely that the division of the nerve into three parts—ophthalmic, maxillary, and mandibular—is topographically represented in the ganglion and at least in the distal part of the sensory root. Whitehead¹⁷ follows this up by demonstrating that in embryos the ganglion consists of three distinct parts each associated with its own topographic division of the nerve, and goes on to show that there is 'no tendency for the fibres of the sensory root to intermingle.' Woollard and Carmichael¹⁸ state more guardedly that 'a study of the ganglion and the central root by anatomical methods has led to the conclusion that there is no complete segregation of fibres in the central root corresponding to epicritic and protopathic functions.'

Reports have also been made on the Vth nerve of animals other than primates. The present writer is not prepared to accept these as having definite bearings on the practical problem of the disposition of the fibres in man.

Dissections of the Vth nerve in man have proved disappointingly inconclusive,

* Since this work was done the writer has seen a paper by Peters¹⁶ describing these same cells in various animals. Peters's publication was in October, 1935, the writer's observation approximately at the time of Peters's publication.



FIG. 99.—Right half of medulla of *Macacus* eleven days after resection of posterior third of sensory root of trigeminal nerve close to pons. Massive degeneration of spinal root of Vth. (Marchi.) ($\times 90$.)

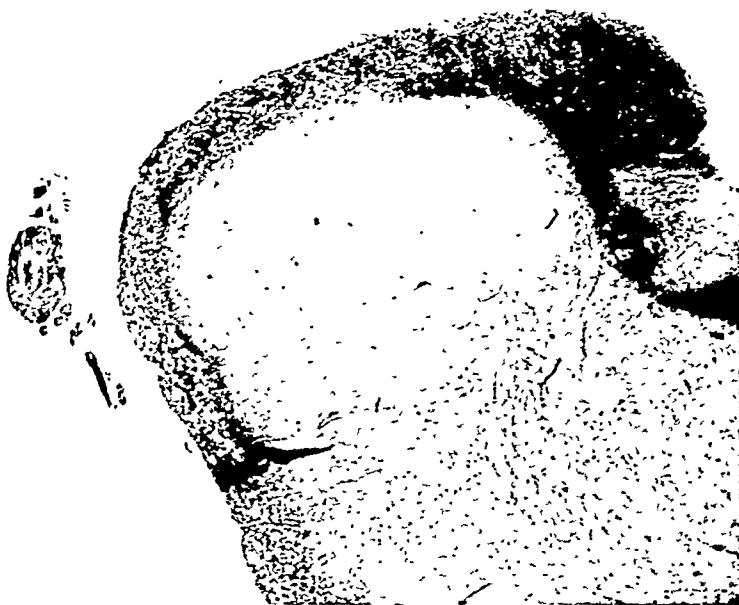


FIG. 100.—Control left side of medulla of *Fig. 99*. (Marchi.) ($\times 90$.)

possibly owing to the failure so far to devise a technique. It is certain that the fibres of the sensory root do intermingle, crossing and recrossing; that fasciculi, jutting off, branch here and there, joining up with one another again, and splitting. The superficial attachment of the nerve to the pons is exceedingly fragile in virtue of the fact that immediately upon entering the pons the fibres break away to such an extent that so far no single convincing and complete dissection has been produced. On the other hand, these difficulties should not be insurmountable, and the imperfect dissections (*Fig. 101*) strongly suggest that the nerve close to the pons is segregated



FIG. 101.—Dissection of sensory root of trigeminal nerve. A, Protopathic fibres to spinal root; b, Fibres going mostly to principal nucleus, b', A few antero-medial fibres to spinal root

into two parts: (1) Outer or posterior fibres which go entirely to the spinal root; and (2) Inner or anterior fibres, some of which go to the spinal root but most to the principal nucleus. It is proposed to follow this side of the work up by devising some method for fixing in situ before dissection. The histological structure of the nerve is also being studied and the results are being reported elsewhere; they should, when complete, offer useful collateral evidence.

SUMMARY

1. A brief history is given of the origin of two methods of approach to the Vth nerve for surgical resection in the treatment of trigeminal neuralgia—namely, Dandy's and Frazier's.

2. Experimental work on monkeys shows that there is in the sensory root of the Vth nerve a crossing and segregation of the fibres into spinal and principal nucleus fibres.

3. This division can properly be interpreted as a functional division into pain and touch fibres.

4. The sensory root of the Vth nerve contains numbers of large sensory nerve-cells, in scattered groups often quite close to the pons. Section of the nerve distal to these as practised by Frazier does not rule out the possibility of regeneration of the fibres.

REFERENCES

- ¹ HORSLEY, *Brit. Med. Jour.*, 1891, pt. 2, 1139.
- ² TIFFANY, *Ann. of Surg.*, 1894, xix, 47.
- ³ FRAZIER, *Philadelphia Med. Jour.*, 1902, x, 594.
- ⁴ DANDY, *Bull. Johns Hopkins Hosp.*, 1925, xxxvi, pt. 2, 105.
- ⁵ STOPFORD, *Sensation and the Sensory Pathway*, 1930. London.
- ⁶ GILLIS, *Jour. Amer. Med. Assoc.*, 1914, lxiii, 1550.
- ⁷ HARRIS, W., *Proc. Roy. Soc. Med. (Neurol. Sect.)*, 1909, ii, 77.
- ⁸ GERARD, M., *Arch. Neurol. and Psychiat.*, 1923, ix, 306.
- ⁹ STOPFORD, *Jour. of Anat.*, 1924-5, lix, pt. 2, 120.
- ¹⁰ GRAY, *Anatomy*, 20th ed., 984.
- ¹¹ GUDDEN, *Allg. Zeits. f. Psychiat.*, 1891, xlvi, 16.
- ¹² RANSON, *Brain*, 1915, xxxviii, 487.
- ¹³ KOCH, *Jour. Comp. Neur. and Psych.*, 1916, xxvi, 541.
- ¹⁴ KAPPERS, *Die vergleichende Anatomie der Nervensystem der Wirbelthiere und des Menschen*, 1920. Haarlem.
- ¹⁵ STOPFORD, *Loc. cit.*
- ¹⁶ PETERS, *Jour. Comp. Neurol.*, 1935, lxii, 349.
- ¹⁷ WHITEHEAD, *Brain*, 1925, xlvi, pt. 1, 458.
- ¹⁸ WOOLLARD AND CARMICHAEL, *Proc. Anat. Soc.*, 1932, lxvii, 204.

RECONSTRUCTION OF THE COMMON BILE-DUCT

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THE children of Israel, when commanded by the Egyptians to make bricks without straw,¹ were in no more difficult position than the modern surgeon who is faced with the task of reconstructing the common bile-duct, particularly when the damage has been so extensive that the right and left hepatic ducts are represented only by two yawning black holes on the under surface of the liver. One of the most difficult technical procedures in the upper abdomen is the reconstruction of a damaged common bile-duct. The purpose of this paper is to describe a method of reconstruction in a specific case, and to discuss the principles which have contributed to a successful issue. So far as can be determined, some of the details herein described are unique.

Edmund Horgan,² in a very complete monograph, *The Reconstruction of the Biliary Tract*, provides 'a review of all the methods that have been employed', and lists, in this work, nearly every conceivable anastomosis between liver, hepatic ducts, cystic duct, gall-bladder, common duct, duodenum, stomach, jejunum, colon, and external fistula.

As Coors³ observes, almost every type of short-circuit has been carried out. The gall-bladder has been anastomosed successfully to stomach, duodenum, jejunum, common duct, and colon. The cystic duct has been anastomosed to stomach, duodenum, jejunum, and colon. The common duct has been anastomosed to stomach, duodenum, and jejunum. The hepatic duct has been anastomosed to stomach, duodenum, and jejunum. Even denuded liver substance has been anastomosed to duodenum and jejunum. End-to-end anastomosis of damaged ducts has been done either with or without the use of the splinting aid of rubber tubing, T tubes, and L tubes. Implantation of an external biliary fistula has been successfully made into the stomach, duodenum, bile-duct, and jejunum.

Plastic reconstruction of portions of the bile-duct has been accomplished both by pedicle flaps from the stomach and longitudinal incisions sutured transversely. Strictures of the ducts have been successfully dilated by means of forceps and bougies. The above-mentioned anastomoses have been accomplished both by direct coaptation and also by the utilization of many ingenious intervening foreign materials, such as Murphy buttons, decalcified bone bobbins, and rubber tubing with clever cuffs upon which are superimposed rubber rings to aid their retention in the ducts for sufficient time to permit complete epithelialization of the newly formed canal.

Nowhere, however, does Horgan describe a procedure which corresponds exactly to that which we were compelled to carry out in this particular case. In the first place, all his operations on hepatic ducts are sufficiently low for the right and left hepatic ducts to have already united to form the common hepatic duct. He does not mention any operation on hepatic ducts which are so short that the right

and left sides are without any extra-hepatic tissue whatever. In the second place, he does not describe a four-way tube such as we devised, to effect the repair of this type of injury.

Since the gradual trend in favour of cholecystectomy as opposed to cholecystostomy, injury to the common duct is no longer rare, in spite of all the efforts to avoid it. There is no arbitrary best method of removing the gall-bladder. Perhaps in most cases where the removal is simple, it is best accomplished from below upward after dividing the cystic duct. In the very obese patient with a deep-seated gall-bladder and liver well up under the costal margin, removal from above downward may favour visualization and so be safer.

In any case, the one underlying principle most likely to safeguard the common duct from mechanical injury, though perhaps not from later fibrosis, is the unyielding insistence on adequate exposure and direct visualization of cystic, common, and hepatic ducts before dividing any duct structure at all.

The history of our patient may be briefly cited. Mrs. E. H., aged 40, gave a history of cholecystitis with biliary colic in 1910. She suffered from typical gall-bladder indigestion with occasional attacks of colic at widely spread intervals from 1910 to 1932. At this time her gall-bladder was removed with considerable difficulty owing to dense adhesions. The cystic duct was dilated to the size of the gall-bladder, and, being filled with large, neatly packed, faceted stones, so resembled the gall-bladder that it was removed completely. A portion of the common duct was either removed or so damaged as to be useless.

For six months following operation the patient was relatively comfortable and showed no external fistula. At the end of that time she suffered a recurrence of pain associated with fever, chills, and jaundice.

After routine pre-operative preparation by transfusion and intravenous calcium chloride, a further operation was carried out for the purpose of re-establishing the continuity of her common bile-duct. At the time of operation she was in only fair general condition. She had lost weight and was deeply jaundiced, with itching.

The operation was technically difficult owing to the dense adhesions present. With patience, however, a clear and adequate exposure was obtained, revealing, to our dismay, two large black holes on the under surface of the liver. Both the right and left hepatic ducts were dilated to about 1 cm. in diameter and there was no extra-hepatic duct tissue. The remnant of the common bile-duct was represented at the other end by a small channel 1 in. long which extended upward from the duodenum in the direction of the under side of the liver.

A catheter was introduced into both right and left lobes of the liver for a distance of about 6 in., and after the intrahepatic duct system was washed thoroughly with saline, a small amount of muddy bile was obtained. Again necessity became the mother of invention, and a large T tube, converted into a four-way tube, was placed in position. The method of modifying the T tube is best shown in *Figs. 102, 103*, and needs no further explanation.

The duodenum, together with the attached stump of common bile-duct, was drawn up as close as possible to the under side of the liver, and in this position was securely fastened around the neck of the four-way tube by means of interrupted sutures, all of which were placed in position before being tightened. The long end of the tube was brought to the surface through a separate stab-wound.

The abdominal wall was closed in layers, and the patient was returned to bed in only fair condition.

We may pause at this point to emphasize the value of a clean exposure of the whole field of operation before proceeding to any active repair operation. The time consumed in clearing the field gently and safely, yet thoroughly, may be much greater than that consumed in the actual repair, but it is time well spent, for by this means only is it possible for an accurate and gentle re-adjustment to be made. Furthermore, the situation is well in hand at all times, and the dangers of bleeding or damage to bowel or other tissue are thus minimized, a consideration of value in a jaundiced patient who bleeds easily.

The post-operative convalescence was decidedly stormy owing to the complication of continuous oozing of blood into the abdominal wall—a result, no doubt, of the jaundiced condition. Repeated transfusions were necessary in order to save the patient's life. Later, when the danger of death from hæmorrhage had been overcome, the convalescence was further complicated by loss of appetite consequent upon loss of bile to the surface.

Loss of appetite and loss of spirit are definite and well-recognized accompaniments to loss of bile, and can at times be cause for real concern. The patient may become depressed and melancholic. This fact again confirms ancient observations that the biliary flow has an influence upon moods. The evil effect of loss

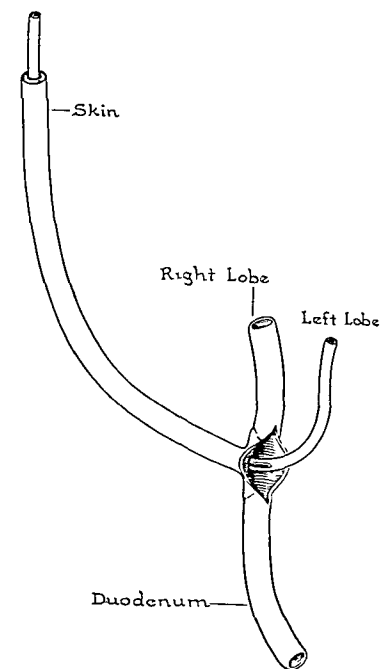


FIG. 102.—T tube converted into a four-way tube, showing the destination of its extremities.

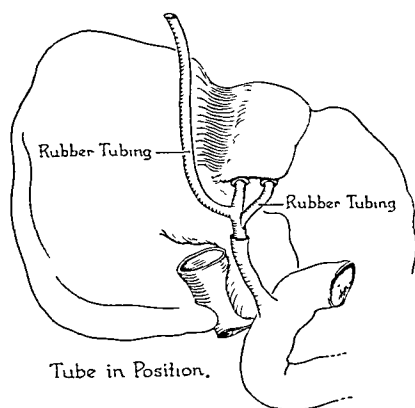
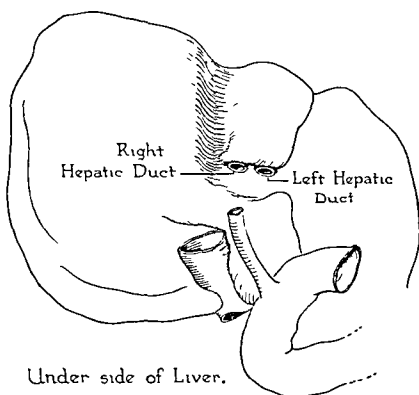


FIG. 103.—Showing the four-way tube fitted into position.

of spirit on both patient and surgeon is neatly circumvented to-day by the simple expedient of catching the patient's own bile in a bottle hung under the bed, and

feeding it back to him daily via the duodenal tube. The new lease of life resulting from the return of bile to the digestive tract has been the turning-point in more than one convalescence.

The four-way tube in this case was, however, kept clear by irrigation, and gradually the discharge to the surface diminished. The patient was then allowed to go home wearing a dressing pad over the end of the tube, which, two hours prior to each meal, was shut off, thereby forcing the bile into the duodenum.

The patient continued at home to gain in health and spirit. Her appetite improved, her urine and bowel movements were normal in colour, and the jaundice disappeared. Accordingly, she was allowed to resume her household duties for a period of eight months, at the end of which time she was re-admitted.

After the injection of thorotrast through the tube, the condition of her biliary system was carefully investigated by means of X rays.

Thorlakson and MacMillan,⁴ by demonstrating the changes which occur, have confirmed the surgical observation that these tubes should remain in position for a very long time. They point out that the dilatation of the intrahepatic biliary tract, as a result of calculus, cicatricial, or neoplastic obstruction, produces a state of affairs within the liver quite comparable to bronchiectasis within the lung, and call this dilatation 'cholangiectasis'. The damming back of bile produces: cylindrical dilatation of the common duct and hepatic ducts; sacculaton of the intrahepatic biliary system; pressure atrophy of the parenchymatous liver cells; interference with the portal circulation; cholangitis; hepatitis; and, finally, fibrosis of the whole liver with impairment of its function.

They show these structural changes radiographically, and demonstrate the improvement which follows prolonged drainage.

The degree of dilatation and damage is said to correspond with the severity of infection and duration of the obstruction. The ratio influences, therefore, the length of time necessary for drainage. If lipiodol or thorotrast is injected through the T tube, some will find its way into the duodenum. Most, however, will pass into the intrahepatic duct system to its finest radicles although there is no obstruction at the duodenum. Failure to visualize the intrahepatic system indicates, therefore, a widespread cholangitis, which will subside following prolonged drainage.

The technique of this investigation is simple: 20 c.c. of thorotrast are injected, plates are made immediately, and again at the end of twenty minutes. Dilatation of the main duct, sacculaton of the lesser ducts, or delay in emptying are indications that the liver and duct system have not recovered. Drainage, then, must be continued. When the liver tree is normal and drainage prompt, the tube may be removed. By this method, it is possible also to prove free flow of bile through the ampulla of Vater. While it is true that others⁵⁻⁸ had previously injected opaque materials into the biliary system, Thorlakson and McMillan were the first to pursue this study in a consistent manner, and to correlate X-ray findings within the tree with the physical condition of the patient. It is a well-known clinical observation that the T tube must be left in for a long time, but we have, in this method of investigation, a means of indicating when the tube may be removed, by the careful measurement of the improvement taking place in the liver.

When it was demonstrated in our patient (*Figs. 104-107*) that there was no undue sacculaton of the biliary tree and that emptying occurred promptly into the

duodenum, we removed the four-way tube by simply pulling it out. The fistula discharged bile for a few days, after which time it closed without any further



FIG. 104.—Injection of 40 c.c. of thorotrast through four-way tube into liver. (Tube should not yet be removed.) Note: (1) Clubbing and cylindrical dilatation of intrahepatic tree; (2) Thorotrast in duodenum proving patency of ampulla; (3) Thorotrast in pancreatic duct.



FIG. 105.—Film taken twenty minutes later than Fig. 104. Note: (1) Still some clubbing and dilatation of intrahepatic tree (2) Poor emptying.



FIG. 106.—Injection of 40 c.c. of thorotrast through four-way tube into the biliary system. (Tube ready to be removed.) Note: (1) Disappearance of clubbing and cylindrical dilatation and more normal tree extending in fine ramifications to edge of liver; (2) Thorotrast in duodenum proving patency of ampulla; (3) Pancreatic duct.



FIG. 107.—Film taken twenty minutes later than Fig. 106. Note: (1) Good emptying of biliary tree.

operative interference. Since that day, over three years ago, she has been in very satisfactory health, showing no jaundice, appetite good, still thin, occasional attacks of nausea and 'biliousness', but no pain, no fever, and no chills.

In conclusion we may mention one or two principles which seem to have been of importance in this case. Undoubtedly, the greatest single factor in the keeping out or getting out of trouble in any difficult surgical procedure is painstaking and adequate exposure. This will safeguard the tissues from mechanical damage, but in the case of the common duct, will not guarantee against later scar stricture nor prevent stricture arising from inflammation or that due to sloughing of part of the wall of the common duct, a consequence which may result from too enthusiastic dissection. If, during cholecystectomy, it is recognized that damage to the common duct occurs, it should be repaired at once, although this does not ensure against later stricture formation.⁹

The type of reconstruction chosen in this case and the means adopted for carrying it out were further factors contributing to the successful outcome. The four-way tubes, improvised and used as illustrated, ensured a free outlet for the bile from both right and left lobes of the liver to the duodenum, the additional limb to the surface being merely a safety-valve. The liver was decompressed and suffered no damage, and the patient was at all times receiving her usual quota of bile so that the digestion did not suffer.

In this particular case the post-operative transfusions were of inestimable value, for by them only was the patient kept alive. We are fully cognizant of the many elaborate tests to foretell hæmorrhage following operation on jaundiced patients, but in spite of all that have been described, there is still no way of predicting which jaundiced patient will bleed post-operatively. The safe rule is to assume that all will bleed and to treat them accordingly, before and after operation.

Furthermore, there is no sure preventive or cure for this most troublesome tendency. The normal and best stimulus for clotting is hæmorrhage, and in most cases oozing stops after a certain time regardless of vigorous or mild efforts on the part of the surgeon. The one and only therapeutic standby which weathers the test of time is whole blood transfusion. The addition of normal blood to the circulation provides something which the patient lacks, and by so doing stimulates clotting. Transfusions administered for this purpose rather than for the purpose of replacing blood-loss need not be large. Repeated small transfusions of 200 c.c. daily are better than fewer, larger ones.

Finally, the use of a T tube, or some such mechanical device about which to build the new duct, is necessary, and this tube must be left in position for many weeks. Its purpose is not merely to act as a mould about which the duct may be fitted, but rather to mould the growth and repair efforts of nature so that there may be a patent channel from both lobes of the liver to the duodenum. When one remembers that around such damaged tissues healing must occur by granulation and scar tissue, it becomes obvious that the supporting tube must remain in position until all fibrous tissue has been laid down, has contracted to its full extent, and has been held in check by the tube. The rigid fibrous channel thus formed must also be lined with epithelium so that the once patent channel may remain open and not heal across. It is not until this stage, which takes from six months to two years, has been reached, that the new duct is safe from further stricture by contracting scar or from obliteration by fusion of its walls, and therefore it is not until this time that the tube may be removed.

During the time that the reconstructed duct is reaching a state of secure equilibrium, a second equally important effect is being accomplished. The damaged

liver and intrahepatic biliary tree is decompressed by virtue of the ready egress provided for the bile, and thus is permitted to recover from any back pressure effects or inflammation. The dilated ducts contract and emptying is prompt, both evidences of good function. These changes may be watched and proven by X rays following injection of thorotrast into the biliary tree.

I wish to acknowledge the indebtedness of both the patient and myself to Dr. Roscoe R. Graham and Dr. James E. Barry, at the time of operation and also during the immediate post-operative difficulties.

REFERENCES

- ¹ *Exodus* v. 6-7.
- ² HORGAN, E., *Reconstruction of the Biliary Tract*, 1932. New York: MacMillan.
- ³ COORS, GILES, "Reconstructive Surgery of the Common Bile-duct", *Southern Med. Jour.*, 1935, xxviii, 351.
- ⁴ THORLAKSON, P. H. T., and McMILLAN, J. C., "Common Duct Obstruction with Lipiodol Studies of Cholangiectasis and the Effects of Prolonged Drainage", *Canad. Med. Assoc. Jour.*, 1934, xxi, 265.
- ⁵ GABRIEL, W. B., "Proof of Patency of Common Bile-duct by Injection of Lipiodol", *Lancet*, 1930, i, 1014.
- ⁶ OVERHOLT, R. H., "Biliary Tract Visualization with Radiopaque Oil", *Surg. Gynecol. and Obst.*, 1931, lii, 92.
- ⁷ GINZBERG, L., and BENJAMIN, E. W., "Lipiodol Studies of Post-operative Biliary Fistulæ", *Ann. of Surg.*, 1930, xci, 233.
- ⁸ JUDD, E. S., and PHILLIPS, J. R., "The Potency of the Biliary Ducts", *Surg. Gynecol. and Obst.*, 1933, lvii, 668.
- ⁹ HUNT, VERNE C., "Reconstruction of the Common Duct", *Surg. Clin. of N. America*, 1934, xiv, 1389.

CONTINUOUS INTRAVENOUS INFUSION

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It is now almost universally appreciated that the intravenous route for administering salines offers a rapid and sure method of increasing the body fluids and the salt content of the tissues, and that this may be a very important part of the treatment of shock, persistent vomiting, and toxic states. The method of continuous drip infusion has been used for many years, but little work has been done to determine the fate of the various substances which may be given in solution. The present work has been undertaken for the purpose of estimating the value of some of these methods. The clinical and biochemical observations made on a series of cases will be published later. This communication is concerned with a description of the apparatus that has been devised and which is now in use in the wards at the General Infirmary at Leeds.

Although from the clinical point of view it is satisfactory to give saline for short periods and at a very slow rate without regard to the temperature of the fluid entering the vein, the addition of a heating mechanism has the very marked advantage that the rate of flow of the fluid can be regulated within wide limits and that in an emergency one or two pints can be introduced into the circulation in ten minutes or a quarter-of-an-hour. In the General Infirmary at Leeds the standard method of giving intravenous salines during the last few years has been by means of a suspended thermos flask and a Murphy's drip. The disadvantages of this method are manifold. The thermos flask can never be adequately sterilized; it has to be changed frequently owing to its small capacity, the apparatus must be dismantled for replenishing, during which time the blood enters the cannula and clots; and there is nothing to call the attention of the nurse to the fact that the flask is nearly empty. The greatest objection to the apparatus, however, is that it fails to achieve the end for which it is specifically designed—that is, to deliver warm saline into the vein. It is not sufficiently realized that the fall of temperature along a rubber tube in such an apparatus is extremely rapid, and that the passage of the fluid through only a few feet of the tube is sufficient to reduce its temperature to that of the room.

Two of the main difficulties have been overcome in the following way: a large reservoir made of glass and holding enough saline for twenty-four hours was graduated so that the volume of its contents could be read off at any time, and the fluid was heated to the required temperature close to the point at which it entered the vein by means of a thermo-regulated water-bath. The temperature of the bath could be adjusted within fairly wide limits. In the early experiments the fluid was passed through a glass spiral immersed in the bath with the result that gases were liberated and air-locking occurred. The spiral was therefore replaced by the glass apparatus shown in *Fig. 108*. The fluid passes through a side tube C into

a wide vertical tube A, where it is heated to the temperature of the bath. From the lower end of this it passes into an outflow tube B, which is of narrow bore, and so on through a length of pressure tubing and a cannula to the patient's vein. As the limb of the tube A is of relatively large capacity, the rate of flow through this part of the apparatus is slow and the fluid is heated to the temperature of the bath before it reaches the limb B. The gases are therefore liberated in the wide limb A and provision is made for their collection in a bulb D above. The gas as it accumulates can be removed at intervals of twenty-four hours through an outlet tube E in the top of D. This has been called an 'infusion air trap', and it is made by Messrs. Reynolds & Branson Ltd., of Leeds. It fits firmly into a cork which holds it in a hole in the top of the water-bath. The dimensions are as follows: the wide limb is 1 in. in external diameter and 6 in. long from the bulb downwards. The

narrow limb is $\frac{1}{4}$ in. in external diameter and 6 in. long, and the bulb itself is 2 in. in diameter. In order to increase its strength a metal ring encircles the two limbs of the tube, and at this point there is wedged between them a small piece of cork. When the trap is in position the inlet C is separated from the hot lid of the water-bath by a sheet of asbestos so that no warming of the fluid takes place until it reaches the limb A of the air trap. Before this precaution was taken air was liberated in the tube between the drip and the trap, and locking occurred. It is very simple to estimate the rate of fall of temperature along that part of the tubing between the source of heat and the patient for different rates of flow and for different temperatures of the bath with a constant room temperature. This is done by piercing the wall of the rubber tube at intervals and inserting thermometers along its length. The rate of flow is conveniently plotted as drops a minute in the Murphy's drip. This information provides the means whereby the temperature of the fluid as it enters the patient's

vein can be estimated within a few degrees in widely varying circumstances.

Fig. 109 shows the method in use at the General Infirmary at Leeds, where the investigations have been carried out during the last two years. The infusion fluid is held in a large graduated flask which has a capacity of eight pints, a metal lid which can easily be removed, and a funnel-like outlet below. This is sterilized and hung on a movable metal stand at the bedside. A Murphy's drip is attached to the outlet by a short length of rubber tubing, all of which are sterilized by boiling. The flask is then filled with the required solution. From the drip a rubber tube passes to the air trap and the whole system is filled with fluid to remove air. Pressure tubing bearing the glass cannula is attached to the exit from the trap. On this piece of tubing is a metal clip so that the system can be cut off from the circulation when adjustments are necessary, as for example the removal of air from the glass bulb. When the apparatus is once assembled it has the great advantage that no attention is required apart from the daily refilling of the flask, and as this does not necessitate any interruption of the flow it is a thing which can be done by any

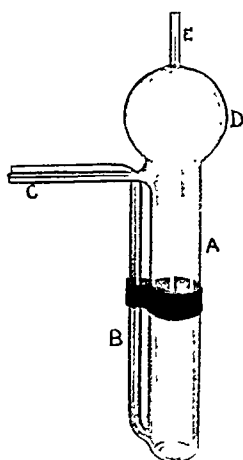


FIG. 108.—The infusion air trap.

nurse who has even an elementary knowledge of asepsis. When salines are to be infused for some days a glass cannula tied into a vein is more satisfactory than a needle, and I am indebted to Mr. R. A. Hall for his suggested use of the cephalic vein in the middle of the upper arm. A cannula tied into this vein and covered with adhesive strapping allows almost full use of the arm and hand.

The thermostatic bath was made by Falk Stadelmann & Co. Ltd., of Leeds. It is a copper bath 12 in. in each dimension, and the heat supply is taken from the electric mains. It is fitted into a special table of which two only of the legs are provided with wheels so that when at rest it is stable, and yet when one end is lifted



FIG. 109.—Patient receiving continuous intravenous infusion. An early case before the use of the cephalic vein was made a routine.

it is easily moved about on the two wheels. When the infusion is once started this apparatus will continue to deliver the fluid to the patient at a known temperature and rate so long as the reservoir is replenished at intervals and so long as the vein remains patent. Owing to the constant and uninterrupted pressure of fluid no reflux of blood into the cannula, with consequent clotting, occurs. After some days, however, the irritation of some fluids causes an aseptic phlebitis with swelling of the vein wall and gradual occlusion of its lumen. When this occurs it is a simple matter to fix the clip on the delivery tube in order to cut off the flow and then to change the cannula and fix it into another vein, usually the corresponding one on the other arm.

The total cost of the whole apparatus can be greatly reduced by replacing the thermostatic bath by a small wooden box containing one or two electric light bulbs and a thermometer. A hole is made in the roof of the box and into this the cork bearing the air trap is fitted. It is, of course, not possible to regulate the temperature as easily as with the more expensive apparatus, and it therefore requires more careful attention.

The following is a brief summary of a typical case history to indicate the value of this form of treatment. Only those details which are relevant to this communication are given, but they are sufficient to indicate the benefits which can be expected and the lines along which the investigations are being conducted.

F. W., male, aged 59 years, was admitted to the General Infirmary at Leeds under the care of Mr. Flint, on Sept. 26, 1934, with a history of fourteen years' epigastric pain half an hour after food, together with flatulence and heartburn. Three years after his first attack a duodenal ulcer had perforated, and following suture of the ulcer he had had relief from symptoms for one year. Since that time there had been repeated attacks of pain of less severity with a recent complaint of vomiting. He was a very emaciated subject for his build, his stomach was distended down to the umbilicus, and he was a typical example of neglected pyloric stenosis. He was treated in bed and given daily stomach washes and continuous intravenous glucose saline infusion for a period of eight days, after which a posterior gastro-enterostomy was performed. During the preliminary treatment his stomach contracted notably, and his general condition improved beyond all recognition, as the following table shows :—

DAY	URINE	INTRAVENOUS SALINE	WEIGHT
	Oz.	Pints	
1	8	nil	7 st. 10 lb.
2	16	4	
3	34	4	
4	52	5½	
5	92	6	
6	110	6½	
7	88	3½	8 st. 7½ lb.
8	88	6	
9	140	3½	

At no time during the treatment did any sugar appear in the urine. The total gain in weight was 11½ lb., due almost entirely to the fluid which had been retained in the tissues. The strength of the glucose solution was 6 per cent, so that the total quantity of glucose metabolized was about 110 gm. a day, taking the average rate of infusion as four pints in the twenty-four hours.

TWO CASES OF GANGLIA IN THE SHEATH OF THE PERONEAL NERVE

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CYSTIC degeneration of a nerve-sheath is a condition which does not appear to be mentioned in text-books of pathology or neurology, but several cases have been recorded in Continental, though none has been found in the English, literature. This condition is associated with sensory changes and paralysis of the affected nerve, and it is interesting that out of 13 recorded cases, the peroneal nerve has been involved on 7 occasions.

The earliest case appears to have been reported by Hartwell,¹ of Boston, in 1901. There was a cyst of the size of a hazel-nut in the median nerve with impaired mobility of the forearm and loss of sensibility to pain and touch. On operation the cyst was found to contain a watery and mucoid substance. Complete recovery followed its evacuation. The writer knows of one somewhat similar case where the median nerve was also involved.

Cysts of the peroneal nerve have been recorded by Sultan,² of Kiel, Zaar,³ and Wadstein.⁴

Etiology.—The frequency with which the peroneal nerve is involved as it winds round the neck of the fibula suggests that trauma plays a part in the causation of the condition, as perhaps does its analogy to the very similar ganglia on tendon-sheaths. Zaar lays emphasis on the great part played by trauma; nevertheless a definite history of injury is not always obtainable.

Symptoms.—In nearly all cases the first symptoms are those of pain in the areas supplied by the affected nerve. The pain varies in intensity and may be accompanied by numbness; after a few weeks the patient notices weakness of the extensor muscles and complains of drop-foot.

Diagnosis.—The patient's chief complaint is usually that of foot-drop. On examination of the leg a swelling can be felt in the course of the peroneal nerve which may be tender. It is cystic in nature, may be lobulated, and tender to touch, extending from the front of the neck of the fibula some distance up the nerve behind the biceps tendon. The swelling is not attached to skin or deeper structures; it is never associated with swellings on other nerves or signs of a generalized neuro-fibromatosis.

Pathology.—The tumour forms a fusiform translucent swelling lying under the sheath of the nerve-trunk and occasionally following one or more of its branches into the muscles of the leg. The tumour is lobulated and lies only partially between the nerve bundles. The wall of the cyst is composed of connective tissue without nervous elements. The contents are a colourless, glairy, jelly-like fluid, exactly similar to that found in ordinary ganglia.

Treatment.—Simple incision of the cyst and expression of its contents can be performed without injury to the nerve. The skin is sutured and heals soundly.

There appears to be no liability to recurrence. In cases of short duration the sensory symptoms disappear early and the paralysis passes in the course of a few months.

CASE REPORTS

Case 1.—Stanley C., aged 13. First seen on Dec. 3, 1929, complaining of pain in the dorsum of the left foot and drop-foot of one month's duration. There was a tender swelling $\frac{1}{2} \times 1$ in. just below the head of the fibula. An X-ray showed no bony changes.

OPERATION (Jan. 17, 1930).—The nerve proximal to the head of the fibula was twice as thick as normal, looked œdematous, and felt hard; there were several small translucent cysts. Below the head of the fibula the peroneal branches were intact, and the muscles responded briskly to electrical stimulation. The anterior tibial nerve was apparently replaced by a swelling as large as a walnut, consisting of many freely communicating cystic cavities. This cyst was incised and evacuated. The cysts higher up were milked down and expressed through the same opening. The incision was then closed.

Aug. 5: Electrical reactions: peronei, normal; anterior tibial group, weak response; tibialis anticus did not appear to respond.

Dec. 2: Complete recovery.

Case 2.—John J., aged 44. A corn warehouseman.

Oct. 14, 1935: History of moderate pain in the left foot and foot-drop for one month. There was no definite history of injury, but his work involved filling heavy sacks of grain which he held between his knees, often resting the outer side of his left knee against another sack for support.

ON EXAMINATION.—Paralysis of muscles supplied by anterior tibial nerve. Over the head of the fibula, on its lateral aspect, there was a cystic swelling of the size of a cherry. The swelling was smooth and rounded, not tender, and doubtfully translucent; not attached to skin or bone, and could be moved slightly from side to side.

All the muscles of the anterior group reacted normally to faradism and galvanism.

OPERATION (Oct. 17).—Exploration of left external popliteal nerve (*Fig. 110*). Thin-walled cyst found in sheath of nerve extending for a short distance into peroneus longus muscle. Nerve-fibres compressed but intact. The cyst contained a jelly-like fluid.

Histological Report on Cyst Wall.—Dense fibrous tissue with a lining of flattened cells. No nervous tissue.

Oct. 30: The patient was discharged with a toe-raising spring fitted to the boot.

Jan. 12, 1936: The paralysis had diminished after five weeks, and the function of the foot was normal by this date.

I am indebted to Mr. S. L. Higgs for permission to publish *Case 1*, which was under his care.

REFERENCES

- ¹ HARTWELL, *Med. and Surg. Jour.*, 1901, No. 24.
- ² SULTAN, C., *Zentralb. f. Chir.*, 1921, No. 27.
- ³ ZAAR, *Ibid.*, 1926, 2551.
- ⁴ WADSTEIN, T., *Acta Orthop. Scand.*, 1931, ii, 221.

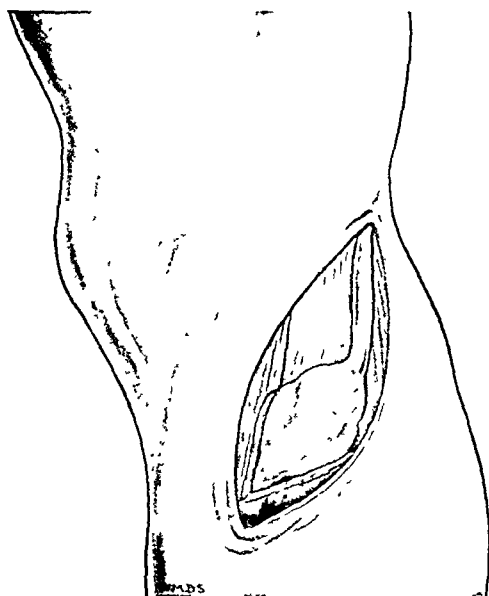


FIG. 110.—*Case 2* View of the outer side of the left knee showing exposure of peroneal nerve with the ganglion lying below the head of the fibula partially in the substance of the peroneus longus, which has been cut.

CYSTIC DISEASE IN SUPERNUMERARY BREASTS

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In 1883 Reclus separated a disease which he called '*maladie kystique de la mamelle*'. Since that time the affection has been described under a variety of names—for example: '*maladie de Reclus*', '*Schimmelbusch's disease*', '*chronic mastitis*', '*chronic cystic mastitis*', '*cystic disease of the breast*', '*chronic interstitial mastitis*', and '*cystadenoma papilliferum*'.

The following case was referred to me for opinion by Mr. H. S. Mehta, M.S., Civil Surgeon, Dhulia, Kandesh, India.

"Mrs. S., was admitted for swelling in both the axillæ. She is a multipara, the last confinement being her third. She is 24 years old. She states that the swellings above referred to were noticed on the 'first day of her last pregnancy',



Fig. 111.—Photograph of patient, showing the two axillary tumours, one on either side.

namely, about a year prior to her admission into the hospital. The swellings increased progressively to their present dimensions. After her delivery she noticed that these swellings used to diminish in size whenever she nursed her child. On examination, both the tumours were found to be soft, fluctuating, freely movable, specially the one in the left axilla. The latter appeared to have no connection with the left breast. Aspiration of both swellings brought out milk. The tumour on the left, being more easily removable, was enucleated and sent for examination." So far Mr. Mehta's notes.

On the evidence to be shortly described, I diagnosed the tumour as cystic disease of a supernumerary breast. Mr. Mehta very kindly supplied me with some photographs along with the excised material. One of these is reproduced

in the text (*Fig. 111*). On examining the sections I requested Mr. Mehta to excise the second tumour and to send it for examination. This request was readily complied with, and about a month later the second tumour arrived with the statement that it appeared to be connected with the right breast by a fascial attachment and that no actual communication with this organ had been discovered. Moreover, no oozing of the contents had occurred on removal.

I cut open each tumour on arrival and collected the contained fluid in test-tubes. In each instance the material had a rancid odour, similar to that of stale condensed milk. The colour was yellowish-white and the consistency that of inspissated milk. Curdy material was present in certain parts of the tumour.

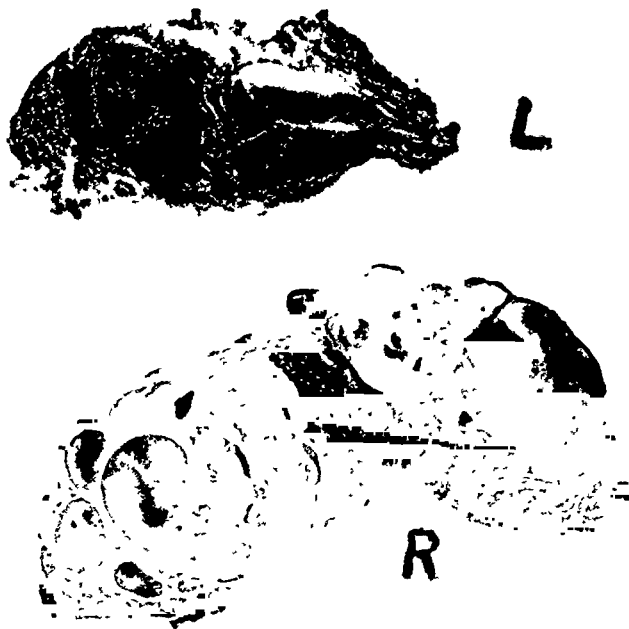


FIG. 112.—Photograph of gross appearance of the tumours submitted for examination.

It cannot be ascertained with any confidence that this was the result of the formal-saline used in fixation, though this does not appear improbable. A drop of the liquid was examined under the microscope with a low power. It was found to consist of fat globules, similar to those seen in ordinary milk. Application of Benedict's test showed the presence of a reducing substance, and a positive test for protein was obtained with Bogg's reagent.

The naked-eye appearance of the two tumours is shown in *Fig. 112*. The illustration ($\frac{3}{4}$ size) shows their exact shape, except for a nipple-like projection in the larger tumour, caused by retraction of the overlying skin. The left tumour is the smaller of the two and is marked L. The right (marked R) is the larger. Both have been enucleated in their entirety and had not been cut into on removal. In the description that follows, the two tumours will be referred to as Tumour L and Tumour R respectively.

Tumour L shows two large cavities on section and many small ones, being honeycombed towards the edges and on the partition between the larger cysts. Sections from the honeycombed areas show lactating acini. In the photomicrograph illustrating these (Fig. 113) one can see a dilated duct with papillary processes projecting into the lumen.

Tumour R, as seen in Fig. 112, is the larger of the two. The cystic dilatations are more numerous and of variable size. In the left half near the middle line, the honeycombed area is filled with a white curdy material. Sections from the less affected areas show lactating acini, but there is greater profusion of dilated ducts, some of which show proliferation of the lining epithelium (quite apart from the effect of oblique section). In some places a well-marked round-cell infiltration is present. In other sections, larger papillomata, to all appearances, severed during cutting are present within the lumen of dilated ducts, while in other places acidophil cells and epithelial debris are present within the lumen of the ducts.



FIG 113—Photomicrograph of tumour of left side

The case is published because a bilateral affection of supernumerary breasts is present in the patient. The interest is further enhanced by the fact that the removed breast tissue does not, as far as I could ascertain, communicate with the adjacent breasts. Moreover, there is no external communication nor even a rudimentary nipple.

The pathology of cystic mastitis is a matter of dispute, and there is great difference of opinion as to its causation. In France it would appear to be common in young women (Letulle), while other authorities hold the view that it occurs on the wrong side of middle age. Keynes's statistics apparently show that in England the disease occurs more frequently in "women nearing the menopause"—that is, between 30 and 40 years.

The theories as to the pathogenesis of this affection are as numerous as the names by which it is known. To mention only a few, it is held by some that the

condition is inflammatory. MacFarland, after an analysis of numerous cases, upholds Warren's theory of "abnormal involution", or "delayed involution" as MacFarland puts it. Schimmelbusch adheres to the neoplastic theory. In his Hunterian Lecture, delivered before the Royal College of Surgeons in England on May 9, 1923, Geoffrey Keynes arrives at the following conclusions, among others:—

"1. The breast is a secretory gland which shows periodical activity from birth to menopause.

"2. The normal non-lactating breast has no outlet through the nipple for the discharge of its secretions. Secretion must therefore be balanced normally by reabsorption.

"3. Chronic mastitis is manifested by dilatation of ducts and acini, accumulation in them of the products of epithelial activity, infiltration with lymphocytes, fibrosis, and epithelial changes. Distribution of all these is very erratic.

"5. Chronic mastitis is not bacterial in origin, toxæmic, or traumatic. Nor is it related to involution changes in the breast.

"6. The cause of chronic mastitis is probably to be found in chemical irritation due to stagnating secretions and epithelial debris. This cannot be proved until the chemical changes have been investigated."

I mention these conclusions in detail, as the case above described would appear, in a measure, to substantiate Keynes's contention. In the first place, the affection is that of supernumerary breasts without any outlet for the resultant secretions. Secondly, the trouble arose during lactation, though one could not ascertain whether any hypertrophy of the additional breast tissue occurred during previous pregnancies. It would appear from the history that the patient did not notice anything previously. Still, it is well known that during lactation some acini may remain dormant. The theory of stagnating secretions would appear to apply particularly to the case under consideration.

BIBLIOGRAPHY

BOYD, *Surgical Pathology*

KEYNES, *Brit. Jour. Surg.*, 1923, xi, 89.

LETULLE, MAURICE, *Anatomie pathologique*.

MACFARLAND, *Surgical Pathology*.

THE SIGNIFICANCE OF LUMBOSACRAL TRANSITIONAL VERTEBRÆ

By G. A. G. MITCHELL,

SURGICAL SPECIALIST FOR THE COUNTY OF CAITHNESS

MAN'S assumption of the erect posture has necessitated many skeletal modifications and these are nowhere more apparent than about the lumbosacral junction. Finality in structure has not yet been attained and developmental abnormalities are abundant, a fact of practical importance, as some anomalies strengthen the spine while others weaken it. The presence of an enlarged transverse process on one or both sides of the last lumbar vertebra is one of the best known of these anomalies, and it is interesting both from the anatomical and clinical view-points. The condition is usually referred to as sacralization or lumbarization, and its occurrence is puzzling unless one possesses a knowledge of the anatomy and morphology of the spinal column.

ANATOMICAL SIGNIFICANCE

The normal formula for the human vertebral column is : C.7, T.12, L.5, S.5, Co.4, but variations from this occur in about 20 per cent of skeletons. The total number of segments seldom varies, however, and as there is no proof that intercalation or excalation of whole segments occurs naturally, any addition of a vertebra to one region must be associated with the loss of a vertebra from another. Numerical variability is greatest at the sacro-coccygeal junction, and usually results in the acquisition of an extra vertebra by the sacral region and a loss of one from the coccygeal region, but variations at the lumbosacral junction are also common, resulting in either elongation or shortening of the lumbar region.

Phylogenetic advances have been accompanied by reduction of segments at both the cranial and caudal ends of the spinal column. In the evolution of the higher primates the lumbar region has become relatively shortened, the process of reduction being brought about by the transformation of lumbar into sacral vertebrae. As a consequence the whole pelvic girdle has migrated in a cranial direction and the number of pre-sacral vertebrae has decreased. According to Rosenberg these phylogenetic advances are partly recapitulated in human ontogeny, because the pelvic girdle is primarily attached to the twenty-sixth vertebra, but subsequently extends forwards on to the twenty-fifth, while at the same time the thirtieth vertebra, primarily sacral, becomes separated from the sacrum to form the first coccygeal vertebra. Rosenberg's conclusions are not accepted by all authorities; indeed the balance of opinion seems to swing more to the opposing view that in man the pelvis is migrating backwards instead of forwards. If the latter opinion is correct, Rosenberg's theory must be incorrect or incomplete. Most anatomists are agreed that it is correct but incomplete, and to complete the theory Keith has suggested that in the human phylum the lumbar region was again lengthened when the plantigrade mode of progression was substituted for the brachiating.

An idea of the tendency to progression or retrogression of the pelvis can be gained by studying the number of pre-sacral vertebræ. The number of cervical vertebræ remains practically constant, and thus any increase or decrease in the pre-sacral vertebræ usually affects the thoracico-lumbar region. The normal formula for these regions in man is T.12, L.5, giving a modal number of seventeen. If the pelvic girdle progresses in a cranial direction, a lumbar vertebra becomes incorporated in the sacrum and the thoracico-lumbar segments are reduced to sixteen, whereas if it recedes caudally the process is reversed and the number of thoracico-lumbar vertebræ is increased to eighteen. The spinal column of man shows less tendency to numerical variations than that of anthropoid apes, and is characterized by the stability of the modal number of seventeen. Todd has observed that the modal number of the gorilla and chimpanzee is also seventeen, of the gibbon eighteen, and of the ourang sixteen. Thus at first sight it appears that the ourang has progressed further than man, but if Keith's theory is correct it has not this significance, since he suggests that man has reached and passed this stage, and that his lumbar region is becoming elongated again. Rosenberg, Todd, and Willis think that in man the tendency is towards reduction in the number of the thoracico-lumbar segments to sixteen. On the other hand Bardeen, Frets, Keith, Dwight, Fischel, and Shore are of the opinion that the pelvis is migrating backwards in man, tending to increase the number of thoracico-lumbar segments to eighteen. If the almost constant cervical element of seven is added, it is clear that the total number of pre-sacral vertebræ oscillates between twenty-three and twenty-five, and the numerical variations are mainly dependent on a reciprocal gain or loss of one vertebra at the lumbosacral junction. Often the process is incomplete and the vertebra gained by the sacral from the lumbar region, or vice versa, fails to assume completely all the characters of its adopted region, and this explains why transitional types of vertebræ are common at the lumbosacral junction. The transitional vertebra may have lumbar characteristics on one side and sacral on the other, and it is either the twenty-fourth or the twenty-fifth of the pre-sacral series. Clinically this condition is usually referred to as 'sacralization of the fifth lumbar vertebra', but if Keith and other observers who maintain that the pelvis in man shows a preponderating tendency to migrate caudally are correct, then the condition is more often a lumbarization of the first sacral than a sacralization of the fifth lumbar vertebra. The tendency for the sacrum to acquire a segment from the coccyx is greater than its tendency to lose one to the lumbar region, and Shore suggests that in the evolutionary sense acquisition of the thirtieth vertebra (normally the first coccygeal) by the sacrum precedes, and perhaps even makes possible, the release of the twenty-fifth vertebra (normally the first sacral) to the lumbar region. By simply looking at an isolated specimen or a radiograph of the lower spine it is impossible to tell whether sacralization or lumbarization is present; the total number of pre-sacral vertebræ must be known before an opinion can be given.

The most striking peculiarity of the transitional vertebra is the appearance of one or both transverse processes, which are greatly enlarged as compared with the normal lumbar type (*Fig. 114*). These processes are really a combination of transverse and costal elements, the costal being the larger. In a normal first sacral vertebra the anterior two-thirds of the ala sacralis represents the costal and the posterior third the transverse process (*Fig. 115*). Even in a normal fifth lumbar

vertebra (*Fig. 115*) the costal elements form a not inconsiderable component of the transverse processes, but when sacralization is present they have undergone a degree of development analogous to that which normally occurs in the upper sacral vertebræ.

The abnormal costo-transverse processes may be large or small, symmetrical or asymmetrical. They may not be enlarged sufficiently to reach either the sacrum or the ilium, but at other times they articulate with the sacrum (*Figs. 114, 116*), or even with the ilium, on one or both sides. Sometimes there is unilateral or bilateral bony union between the costo-transverse processes and the sacrum (*Figs. 117, 118*), though the processes are often asymmetrical, and the vertebral bodies and arches



FIG 114—A lumbosacral transitional vertebra with bilateral costo-transverse articulations. It is impossible to tell whether this is an example of lumbarization or of sacralization as the rest of the vertebral column was not available for examination.



FIG 115—The whitened portions indicate the costal elements in the last lumbar and first sacral vertebrae.

may remain partly or completely unfused. Absolute symmetry is uncommon. In about 50 per cent the transitional vertebra shows lumbar characteristics on one side and sacral on the other (*Fig. 117*), the remainder showing anomalous developments on both sides (*Fig. 114*). The body of the transitional vertebra is usually wedge-shaped from side to side, being wider on the side with the larger costo-transverse process. The sacral auricular surface may encroach on the transitional vertebra (*Fig. 119*). If the twenty-fourth vertebra (normally the fifth lumbar) becomes united to the sacrum by bilateral bony union, the process of sacralization



FIG. 116.—Unilateral incomplete lumbarization.



FIG. 117.—Unilateral complete sacralization.



FIG. 118.—A six-piece sacrum.

is said to be complete. This may result in a six-piece sacrum (*Fig. 118*), unless a segment is acquired by the coccyx from the sacrum, thus maintaining a five-piece sacrum. Lumbarization, by partially or completely releasing the first sacral vertebra to the lumbar region, would produce a four-piece sacrum were it not for the fact that the five segments of the sacrum are usually maintained by the acquisition of the first coccygeal segment. Four-piece sacra are rare, only occurring in about 1·2 per cent of European skeletons (*Le Double*).

The frequency with which lumbosacral transitional vertebræ are to be expected varies in different races. *Wetzel* found that they were present in 18 per cent of Australian aboriginals; *Shore* estimated the percentage in Bantu natives to be 4·8;



Fig. 119.—Examples of encroachment of the sacral auricular surfaces on lumbosacral transitional vertebræ.

Toyoda discovered that they were present in 5·8 per cent of Japanese spines; *Brailsford*, from radiographic studies, gives the percentage as 8·1 for natives of Britain; and *Keith* states that “in a series of one hundred human skeletons it will be found that some three or four will show a headward sacralization, while six or eight show a tailward movement.”

CLINICAL SIGNIFICANCE

Should complete sacralization of a fifth lumbar vertebra have occurred, the lumbosacral junction is strengthened, and this is true even if the bony union is present only on one side. Complete lumbarization of the first sacral vertebra

lengthens the lumbar portion of the spine, thus allowing slightly increased mobility, but as mobility and strength are opposing virtues the lumbosacral junction is weakened. If two well-formed lumbosacral transverse articulations are present the junction is strengthened, but trouble may arise from the liability of the abnormal joints to arthritic changes. In the other incomplete types there is little doubt that the enlarged costo-transverse processes are occasionally the cause of low back pain and other symptoms. Jones and Lovett say they are rarely responsible for symptoms, and Verrall agrees, but says symptoms are severe when they do occur. Léri says they seldom cause pain, but Mennell in this country and the majority of American writers believe that they are capable of causing symptoms. A few deny that they ever cause symptoms and attempt to justify their contention as follows:

1. The anomaly has been present from birth, yet no symptoms may develop till adult life.

2. The condition may be found during routine examination in patients with no backache.

3. Many who have been operated upon and had this 'cause' of backache removed have not been relieved.

These contentions are easy to refute:—

1. The process at birth is relatively small and partly cartilaginous, and does not reach its full development or become completely ossified till early adult life. Obviously a small, partly cartilaginous process is less liable to cause trouble than a larger, completely ossified structure.

2. Other congenital anomalies, e.g., cervical ribs and spina bifida, may be found by chance during a routine examination for some other condition, yet no one doubts that these anomalies occasionally produce symptoms.

3. It is true that the removal of an enlarged costo-transverse process does not always cure low back pain; but that only proves that the diagnosis and treatment were wrong in that particular instance. The fact that appendicectomy after a misdiagnosis of appendicitis does not cure the patient is no reason for denying that the appendix ever causes pain.

There is a sufficient body of evidence to prove that these enlarged processes are potential and sometimes actual causes of low back pain. They may produce symptoms in various ways:—

1. If the process is not large enough to articulate with the sacrum, lateral bending movements may cause it to impinge on the sacrum, thus nipping the intervening soft tissues and bruising the periosteum.

2. In unilateral cases the impingement of the process on the sacrum or ilium may provide a fulcrum for the spinal column, which then acts as a lever of the second class tending to prise apart the lumbosacral transitional vertebra from the sacrum, straining or tearing the connecting ligaments (*Fig. 120*). It is obvious that the ligaments on the side opposite to the abnormal process will suffer most, and this explains the seeming paradox that the symptoms and signs are sometimes worse on the side with the normal process. Violent lateral bending may cause acute lumbosacral strain, while ordinary lateral bending movements may produce minor repeated ligamentous strains which have a cumulative effect. This type of strain is distinguished by certain writers from other cases of lumbosacral strain by calling it 'lumbosacral transverse strain,' but there is no advantage in this, for it is highly improbable that the ligamentous fibres surrounding the abnormal costo-transverse articulation suffer alone.

3. An adventitious bursa may form between the enlarged process and the sacrum or ilium and become inflamed owing to repeated small injuries.

4. If the process articulates with the sacrum or ilium, a so-called lumbosacral transverse articulation is formed. The articular cartilages of the joint are often thin and therefore not very efficient as shock absorbers, thus lessening the resistance of the joint to traumatic insults. Repeated minor injuries such as are produced by lateral bending, heavy lifts, etc., initiate arthritic changes more readily under these circumstances.

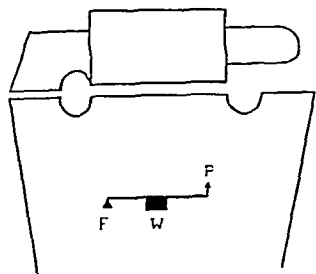


FIG. 120—A diagram to illustrate how levering effects may be produced by unilateral incomplete lumbarization or sacralization.

5. In some cases associated peri-arthritic changes may involve adjacent nerves. Ecchondroses or osteophytes may develop around the margins of the abnormal articulations, encroach on the intervertebral foramina, and press on the contained nerves. In sacralization the fifth lumbar nerve is liable to be involved, while in lumbarization the first sacral nerve is the one liable to suffer.

6. Bauman thinks that the part of the fourth lumbar nerve which helps to form the lumbosacral trunk may be stretched as it passes over the enlarged costo-transverse process, particularly during spinal extension, and that this may give rise to radiating nerve pains.

SYMPTOMS AND SIGNS

Symptoms.—Symptoms due to lumbarization or sacralization often manifest themselves during the third decade, and in this respect the clinical history resembles that of many cases with cervical ribs; this is interesting when one considers that the symptom-producing costo-transverse process is mainly formed by an abnormal development of the costal element. The symptoms may first appear after injury (particularly a fall on the flat of the back), illness, unaccustomed activity, or working in a cramped or unusual position, while anything upsetting the normal posture or gait and throwing more strain on the side with the abnormal process, may initiate the trouble. One patient, for example, developed low back pain due to an incomplete right unilateral lumbarization after injuring a cartilage in his left knee, which led to recurrent attacks of synovitis and caused him to throw more weight on the side with the lumbarization. Verrall remarks on the intermittency of the symptoms; this may be due to recurrent inflammation of an adventitious bursa, recurrent arthritis in a lumbosacral transverse joint, or occasional nipping of tissues between the sacrum and the process.

Low back pain is the most common complaint, and it is fairly well localized over the lumbosacral region. In some cases it is very severe and may completely incapacitate the individual. In others it takes the form of an indefinite dull ache. Like most other forms of low back pain it is extremely intractable at times, and arouses the usual suspicion of neurosis or malingering. Lower spinal movements, walking, coughing, and abdominal straining increase the pain, while lifting weights and bending become difficult or impossible. Owing to the possibility of involvement of the lower lumbar or first sacral nerves, pains in their cutaneous areas of

distribution are not rare, occurring along the outer side of the leg and in the foot. Pains along the posterior aspect of the thigh are less common. In cases with definite nerve irritation or compression the patient may complain of weakness or unsteadiness of the leg on the affected side. Any nerve symptoms and signs in sacralization are practically identical with those which may occur in lumbosacral strain, whereas in lumbarization any nerve features present resemble more closely those which may occur in sacro-iliac strain.

Children rarely have symptoms which could be ascribed to lumbarization or sacralization, although a child is occasionally seen with symptoms suggestive of hip-joint or genito-urinary disease in whom the only discoverable abnormality is lumbarization or sacralization.

Signs.—The paucity of definite objective findings is sometimes surprising in a patient with severe symptoms, another feature which often arouses suspicions of neurosis or malingering. The lumbar region of the spine may appear unduly long if lumbarization is present, while in sacralization this region is shorter than normal and the ilio-costal space is diminished. In patients with severe unilateral trouble a scoliotic deformity away from the affected side may be found. On the other hand, cases have been recorded where the development of a scoliosis due to some other condition caused impingement of an enlarged costo-transverse process on the sacrum or ilium and so produced low back pain in a patient previously free from this complaint.

In most cases localized tenderness can be elicited on pressure over the lumbosacral region. In unilateral cases this tenderness may be more marked over the anomalous process, but in other cases, for reasons already explained, the maximum tenderness may be present on the side showing no abnormality. Movements towards the affected side are limited and painful, rotation in particular being performed with difficulty. Flexion at the lumbosacral junction is diminished, and the majority are unable to touch their toes while keeping the knees straight: even when the lumbar spine is flexed as completely as possible it only becomes flat and does not show the usual backward convexity.

Spasm of the lower spinal muscles, slight tenderness over the sciatic nerve, and cutaneous hyperæsthesia along the outer side of the leg and in the foot are present in all except mild cases, while in a few chronic cases definite wasting of the leg muscles or diminution of the tendo Achillis jerk on the affected side can be distinguished.

RADIOGRAPHIC APPEARANCES

In a child the enlarged costo-transverse process bends downwards towards the sacrum, and if the condition is bilateral an appearance resembling a flattened arch is produced (*Fig. 121*). Rarely distinct evidence of the composite nature of the process is afforded by the appearance of a slight gap or foramen between the transverse and costal elements which go to form the enlarged costo-transverse process. As the person grows older the process increases in size, and in bilateral cases a 'butterfly-wing' shadow which is characteristic is cast on antero-posterior films. Radiographs show whether the process is fused completely or incompletely with the sacrum, or whether unilateral or bilateral lumbosacral transverse articulations are present. In the latter event X rays reveal if arthritic changes have occurred (*Fig. 122*). Unless stereoscopic views are taken, it is easy to imagine that the enlarged

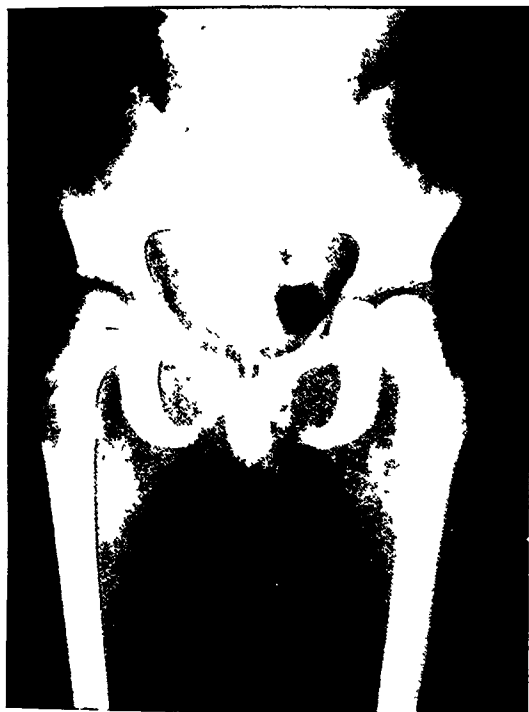


FIG 121—A lumbosacral transitional vertebra in a child aged eight years.



FIG 122—A lumbosacral transitional vertebra with bilateral costo-transverse articulations, one of which shows arthritic changes

process is impinging on the ilium. Actually that is unusual, although in rare instances a joint exists between the two, or the ilium may show signs of erosion through recurrent attrition by an impinging process.

The radiographic demonstration of lumbarization or sacralization in a patient suffering from low back pain does not necessarily indicate that this developmental anomaly is the cause of the trouble. There is often a temptation to attribute the symptoms to the abnormality because diagnosis of low back conditions is never easy, and one is inclined to be satisfied if any possible cause is discovered. But the process should not be incriminated until all other causes have been considered and excluded.

TREATMENT

Conservative Measures.—These should be adopted in the first instance for all cases of lumbarization or sacralization producing symptoms, and the treatment resembles that usually advocated for lumbosacral strain.

If the low back pain is acute, rest in bed is necessary in the early stages, and any period from two to six weeks, depending on the acuteness of the case, is not considered excessive. The patient should lie on a firm bed, with soft pillows under the knees and lumbar spine. During the early stages sedatives may be necessary, and hot fomentations or cold compresses are often soothing. Massage and other physiotherapeutic measures, if employed with skill and discretion, relieve discomfort and stiffness and expedite repair. Thus ultra-violet and infra-red radiation, radiant heat, etc., have been found to give good results in the relief of pain, and at a later date ionization or diathermy may be employed with benefit. Graduated passive and active movements should be begun not later than the end of the first week, and should be continued cautiously till full movements are allowed in the sitting or recumbent position by the end of two to four weeks, the time factor depending on the acuteness of the case. These movements prevent the formation of peri-articular adhesions which are often the cause of persistent pain and disability. Incorrect posture should be corrected, and if it is due to any remediable cause this must be treated. In the severer cases the patient should be provided with a plaster jacket or a properly fitted back-brace when he is allowed up. In females strong specially-made corsets often provide sufficient support and are much more acceptable than a brace. The corsets or back-brace should be worn during the day for three months, but the prolonged use of a brace should be discouraged unless in exceptional cases.

Operative Treatment.—When conservative measures have been faithfully applied and have failed to cure the patient, operative treatment must be considered, but only a small proportion of cases require surgical intervention. Manipulation, which is often so valuable in relieving persistent or recurrent pain following lumbosacral strain, is of doubtful value in lumbarization and sacralization, as the forced movement may aggravate the condition; this is readily understandable when one considers the mechanism of pain production in these cases. Various operations have been suggested, most of which have aimed at the removal of the offending bony process, and the fact that their performance has been attended with success in many cases is strong confirmation of the correctness of the diagnosis. A few operators have found that removal of the outer part of the enlarged costo-transverse process is all that is necessary. From this they argue that the pain must have been

caused by impingement of the process on the sacrum or ilium, or by the levering effect thereby induced, and not through any pressure or tension on nerves. The offending process may be approached from behind either by splitting the erector spinæ or by working down along its outer side. This operation is not easy and hæmorrhage may be troublesome. Verrall has suggested a better method. He cuts a window in the ilium opposite the enlarged process, and through this he removes as much of the process as necessary, a method which has the advantage of removing the part of the ilium on which the enlarged process may impinge, in addition to the abnormal process itself. Other surgeons state that removal of the process tends to weaken the lumbosacral region, and argue that as nature is trying unsuccessfully to fuse the parts the aim of the surgeon should be to assist nature by performing a lumbosacral fusion operation. Although this argument is based on incomplete knowledge, the performance of the operation has given good results in many cases.

A problem arises if this type of congenital anomaly is found by chance in a radiograph. Possibly the best thing is to say nothing about it to the patient, as in many cases it never causes any trouble. Exceptions might be made to this rule in the case of people who perform extremely arduous work, or athletes who indulge in vigorous sports which markedly increase the strains and stresses borne at the lumbosacral junction. The former should be advised to find lighter work if they can possibly do so, while the latter should be counselled to avoid strenuous games.

In certain American works the spine of every new workman is X-rayed as a routine, and if lumbarization, sacralization, or other congenital defects of the lumbosacral region are found which are likely to lead to subsequent trouble, he is not put in a department requiring severe physical exertion. This type of examination, if performed regularly in Britain, would prevent a great number of the medico-legal difficulties which are prone to arise in cases of low back pain and persistent disability.

BIBLIOGRAPHY*

- ABADIE, J., *Bull. et Mém. Soc. nat. de Chir.*, 1924, l, 1054.
 ADAMS, Z. B., *Amer. Jour. Orthop. Surg.*, 1910-11, viii, 229.
 ALESSANDRI, P., *Riv. di Clin. Med.*, 1927, xxviii, 96.
 ANGEL, P. and SENCET, L., *Jour. de l'Anat. et Physiol.*, 1902, xxxviii, 217.
 ANDERSON, E., *Forts. a. d. Geb. d. Röntgenstrahlen*, 1926, xxxiv, 491.
 AYERS, C. E., *New Eng. Jour. Med.*, 1929, cc, 592.
 BANKART, A. S. B., *Brit. Med. Jour.*, 1921, i, 587, 636.
 BARDEEN, C. R., *Anat. Anzeiger*, 1904, xxv, 497.
 BARDEEN, C. R., *Amer. Jour. Anat.*, 1905, iv, 265.
 BASSET, A., *Bull. et Mém. Soc. nat. de Chir.*, 1925, li, 1078.
 BAUMAN, G. I., *Jour. Bone and Joint Surg.*, 1924, vi, 909.
 BAUMAN, G. I., *Ibid.*, 1923, v, 579.
 BAUR, *Jour. of Morph.*, 1890-91, iv, 331.
 BEADLE, O. A., *The Intervertebral Discs*, 1931, (*Med. Research Council Special Report Series No. 161*).
 BENASSI, G., *Anthropologie*, 1927, xxxvii, 125.
 BONNOIT, A., *Lyon Chir.*, 1921, xviii, 445.
 BERRY, J. M., *Arch of Surg.*, 1925, xi, 883.
 BEST, R. R., *Surg. Gynecol. and Obst.*, 1927, xlv, 485.
 BILLINGTON, R. W., WILLIS, T. A., and O'REILLY, A., *Jour. Bone and Joint Surg.*, 1928, x, 290.
 BIRMINGHAM, A., *Jour. Anat. and Physiol.*, 1891, xxv, 526.

* This bibliography is not exhaustive, but contains the majority of the more important contributions to the problem.

- BOHART, W. H., *Jour. Amer. Med. Assoc.*, 1929, xcii, 698.
 BOWKER, C., *Med. Jour. of Australia*, 1927, ii, 808.
 BRACALONI, E., *Radiol. med.*, 1924, xi, 661.
 BRACKETT, E. G., *Jour. Bone and Joint Surg.*, 1930, xii, 325.
 BRACKETT, E. G., BAER, W. S., and RUGH, J. T., *Jour. Orthop. Surg.*, 1921, iii, 507.
 BRAILSFORD, J. F., *Brit. Jour. Surg.*, 1929, xvi, 562.
 BRASH, J. C., *Jour. Anat. and Physiol.*, 1914-15, xlix, 243.
 BROWN, L. T., *Jour. Bone and Joint Surg.*, 1932, xiv, 157.
 BUSTOS, F. M., *Semana méd.*, 1926, i, 144.
 CAREY, E. J., *Radiology*, 1927, ix, 219.
 CHANDLER, F. A., *Jour. Amer. Med. Assoc.*, 1929, xciii, 1447.
 COCHRANE, W. A., *Brit. Med. Jour.*, 1928, ii, 696.
 DWIGHT, T., *Anat. Anzeiger*, 1901, xix, 321, 337.
 DWIGHT, T., *Ibid.*, 1906, xxviii, 33, 96.
 FISCHER, A., *Anat. Hefte*, 1906, xxxi, 459.
 FRETZ, G. P., *Morphol. Jahrb.*, 1911, xliii, 449.
 FREY, H., *Zentralb. f. Chir.*, 1930, lvii, 2898.
 GHORMLEY, R. K., *Proc. Meet. Mayo Clinic*, 1931, vi, 112.
 GHORMLEY, R. K., *Collected Papers of the Mayo Clinic and Mayo Foundation*, 1930, xxi, 670.
 GOLDTHWAIT, J. E., *Jour. Orthop. Surg.*, 1920, ii, 416.
 HANSON, R., *Acta chir. Scand.*, 1926, lx, 309.
 HEISE, H., *Deut. Zeits. f. Chir.*, 1930, ccxxvii, 349.
 HENDERSON, R. B., *Brit. Med. Jour.*, 1925, i, 922.
 HIBBS, R. A., and SWIFT, W. E., *Surg. Gynecol. and Obst.*, 1929, xlviii, 604.
 JONES, R., and LOVETT, R. W., *Orthopædic Surgery*, 1923. London: Oxford Medical Publications.
 KEITH, A., *Brit. Med. Jour.*, 1923, i, 451, 499, 545, 587, 624, 669.
 KLEINBERG, S., *Med. Jour. and Record*, 1929, cxxx, 133.
 LACKUM, H. L. VON, *Jour. Amer. Med. Assoc.*, 1924, lxxxii, 1109.
 LE DOUBLE, A. F., *Gaz. méd. du Centre*, 1912, xvii, 29.
 LÉRI, A., *Jour. méd. franc.*, 1924, xiii, 265.
 LÉRI, A., *Presse méd.*, 1925, xxxiii, 1681.
 LITTLEJOHN, C. W. B., *Jour. Coll. Surg. of Australasia*, 1930, iii, 201.
 MACKY, K. S., *Med. Jour. of Australia*, 1927, Supp. x, 303.
 MARTY, G., *Jour. Amer. Med. Assoc.*, 1924, lxxxiii, 1297.
 MENNELL, J., *Backache*, 1935. London: J. & A. Churchill.
 MEYNADIER, E., *Thèse de Paris*, 1923.
 MILLS, N., *Boston Med. and Surg. Jour.*, 1921, clxxxiv, 659.
 MILTNER, L. J., and LOWENDORF, C. S., *Jour. Bone and Joint Surg.*, 1931, xiii, 16.
 MITCHELL, G. A. G., *Ibid.* 1934, xvi, 233.
 MITCHELL, G. A. G., *Lancet*, 1936, i, 75.
 MOORE, B. H., *Jour. Bone and Joint Surg.*, 1923, v, 212.
 MOORE, B. H., *Ibid.*, 1925, vii, 271.
 MÜLLER, A., *Münch. med. Woch.*, 1928, lxxv, 695.
 NISEGGI, C. H., *Semana méd.*, 1929, ii, 456.
 NUTTER, J. A., *Canad. Med. Assoc. Jour.*, 1925, xv, 1055.
 O'FERRALL, J. T., *Jour. Amer. Med. Assoc.*, 1928, xci, 532.
 O'REILLY, A., *Jour. Orthop. Surg.*, 1921, iii, 171.
 O'REILLY, A., *Jour. Amer. Med. Assoc.*, 1927, lxxxix, 1128.
 OSGOOD, R. B., *Boston Med. and Surg. Jour.*, 1923, clxxxix, 1059.
 OSGOOD, R. B., *Jour. Bone and Joint Surg.*, 1927, ix, 667.
 PYLE, E., *New Eng. Jour. Med.*, 1931, cciv, 1083.
 RICH, E. A., *Jour. Bone and Joint Surg.* 1928, x, 415.
 ROHRBACH, A., *Münch. med. Woch.*, 1929, lxxvi, 592.
 ROSENBERG, E., *Morphol. Jahrb.*, 1907, xxxvi, 609.
 RUGH, J. T., *Jour. Bone and Joint Surg.*, 1923, v, 235.
 RYERSON, E. W., *Ibid.*, 1932, xiv, 154.
 SCHAUFFER, R. M., *Jour. Amer. Med. Assoc.*, 1930, xcv, 1717.
 SHORE, L. R., *Jour. Anat.*, 1930, lxiv, 206.
 SMITH-PETERSEN, M. N., *Jour. Bone and Joint Surg.*, 1924, vi, 819.
 STRATHY, G. S., *Med. Jour. of Australia*, 1925, ii, 68.
 TOYODA, J., *Folia anat. japon.*, 1927, v, 37.
 VERRALL, P. J., *Brit. Med. Jour.*, 1924, ii, 798.
 WARNER, F., *Internat. Jour. Surg.*, 1925, xxxviii, 179.
 WILLIS, T. A., *Amer. Jour. Surg.*, 1929, vi, 163.

THREE CASES OF FRACTURE RESULTING FROM ELECTRIC SHOCK

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FRACTURES resulting from electric shock do not appear to be generally recognized.

Cases in the Literature.—Jellinek⁴ described two instances of bony trauma in men who had sustained electric shocks from lamps without suffering any mechanical violence.* The first, who experienced severe pain in the shoulder, showed radiologically a fissure 1 cm. long in the head of the humerus. The second patient, who had similar severe pain, committed suicide the day after the injury, and at autopsy the head of the humerus was found divided into numerous fragments.

Martin⁶ referred to a man in whom, after contact with a current at 200 volts, it was found that the head of the right humerus "had been knocked off at the anatomical neck" and "had been displaced backwards and completely turned round". It is not stated whether mechanical violence occurred.

Frühmann³ reported the case of a woman, aged 34, who received an electric shock from a lamp without sustaining any mechanical violence. A radiograph showed forward and downward dislocation of the shoulder with avulsion of the greater tuberosity. During a careful attempt, under ethyl chloride anæsthesia, to reduce the dislocation by Kocher's method a further fracture was produced (site not stated).

Simpson-Smith⁸ described the case of a male patient, aged 41, who grasped the terminals of conductors of a 240 volt alternating current in either hand, contact being maintained for three-quarters of a minute. The head of each humerus was dislocated backwards; the left greater tuberosity was fractured, and the upper end of the right humerus was comminuted, the mushroomed head lying free.

Palugyay⁷ described spontaneous fracture of the proximal phalanx of the third finger of a boy, aged 12, occurring on the fifty-fifth day after prolonged (? several minutes) contact with a wire carrying direct current at 110 volts. Other changes—Sudek's atrophy, periosteal new bone formation, and sequestration of necrotic fragments—were present in this and a similar case.

Ziemke¹⁰ described fractures of the skull and of the middle of the humeral shaft, the bone in the latter region being found at autopsy to be so brittle that it broke like glass. The current in this instance, however, was one of 3000 volts; there was extensive charring of the soft tissues and bone, so that heat may have been a factor in the production of the abnormal brittleness. Mechanical violence also was a complication.

* The term 'mechanical violence' is used in the restricted sense of that of blows from falling and the like; it does not comprehend intrinsic violence such as that of uncontrolled muscular contraction.

Blencke² stated, without giving details, that he knew of a case in which radiographs showed distinct fissures in the distal end of the shaft of the radius after the entry of a high-tension current through the finger-tips; complaint was made of local pain.

The same author² and Wagner⁹ describe a case of Kienböck's malacia of the semilunar bone following an electric shock from a "high-tension" current. A stone-breaker, aged 26 according to Blencke or 30 according to Wagner, suffered electric burns of three fingers from contact with the switch of a lift; he fell and lost consciousness. The right wrist became swollen and was still so two years later when it was X-rayed on account of pain experienced during hard work. The radiographs revealed the presence of Kienböck's malacia of the semilunar bone, which was ascribed to the passage of the current, though clearly it might also be ascribed to the fall at the time of the shock or be considered to have no relationship to it. The case is included here because Kienböck's disease is sometimes considered to imply a crush fracture of the semilunar bone.*

Fractures are familiar in injuries from lightning. Jex-Blake gives a review of this subject.

CASE REPORTS

Three cases, all affecting the upper end of the humerus, have been observed by the author.

Case 1.—Crush Fracture of the Head of the Humerus.†

A man aged 48, a hairdresser, complained of pain in and stiffness of the right shoulder. Nine and a half weeks previously, when holding a vibro-massage machine in his right hand, he was reaching with his left to pick something off the marble slab of a sink when he received a shock from a 50 cycles alternating current of 230 volts. His hands were dry; he does not think he touched a tap; the soles of his shoes were of crêpe rubber and he was standing on linoleum. He was unable to drop the machine. He felt agonizing pain all through him, and as though his right arm were being pulled out of his body. He made facial contortions but did not cry out. He felt dazed, but did not fall. A witness estimated the time which elapsed before the current was turned off at about five seconds. On the next day, or the following, he became bruised from above the right shoulder to below the right elbow. About five weeks after the injury he received massage, movements, and radiant heat three times weekly for a fortnight without improvement. He had suffered from no previous disability or injury; there was no history of any fall involving the shoulder.

On Examination.—Nine and a half weeks after the injury, the right shoulder exhibited a negligible range of movement and attempts were painful; the scapular muscles were wasted.

X-ray Examination.—A radiograph (*Fig. 123*) showed a crush fracture of the head of the humerus.

Treatment by graduated manipulations under anæsthesia, massage, movements, and radiant heat resulted in slight improvement only.

* The histological appearances of Kienböck's disease cannot be explained on a basis of fracture alone. Among a number examined microscopically, the author has seen unmistakable evidence of fracture in one, but it was impossible to say whether this had preceded or followed the more general changes.

† This case has been shown by a resident officer before the Neurological Section of the Royal Society of Medicine, and an account, varying in certain details from that here given, appeared in the *Proceedings*.¹



FIG. 123.—Case 1. Crush fracture of head of humerus.



FIG. 124.—Case 2.—Avulsion and fracture of the greater tuberosity of the humerus.

Case 2.—Avulsion and Fracture of the Greater Tuberosity of the Humerus.

A housewife, aged 62, complained of pain in the right shoulder and disability of the right upper limb. The previous day, when carrying an electric kettle, she received a shock from a 50 cycles alternating current of 230 volts and was unable to drop the kettle. She felt a sudden terrible pain all over as if her arms were being dragged off. She felt very distressed and that she was about to die. She was able to scream and hearing was not affected. After an interval which she could not calculate she lost consciousness and fell, with the result, it seems, that the kettle became released or disconnected. There is no recollection of the fall. During the night the whole upper arm became much swollen. She does not know whether her hands were wet or dry at the time of the accident or what shoes she was wearing; the floor covering was linoleum.

On Examination.—Considerable limitation of movement of the right shoulder was found: the range of abduction was about 30° , scarcely any external rotation was possible, and internal rotation was limited by about 20° . There was no pronounced tenderness.

X-ray Examination.—A radiograph (Fig. 124) showed avulsion and fracture of the greater tuberosity of the right humerus.

Treatment.—In view of her age, treatment was confined to early massage, movements with exercises, and radiant heat.

Progress.—The fracture subsequently united, but, with the exception of a range of abduction of about 45° , no improvement in movement was found five months after the injury. The shoulder was nevertheless reasonably comfortable.

Case 3.—Crush (Impression) Fracture of the Head of the Humerus.

A man aged 47, a shorthand-typist, complained of pain in and stiffness of the right shoulder. Eight weeks previously, when in the loft of his house with a metal standard lamp alight in his left hand, he stumbled and touched a water tank with his right hand to steady himself. He immediately received an electric shock, and was unable to release the edge of the tank from his grasp. He experienced a 'terrible shaking sensation'; and, though he tried to shout, was unable to do so. He lost consciousness after an interval which he could not estimate. When he regained consciousness, contact had ceased, and he now experienced severe pain in the right shoulder and was unable to use the right arm; his chin and the left side of his face were cut. He was in the loft for less than five minutes altogether, but it is unknown for what fraction of this time he remained in circuit. The current was at 240 volts and alternating at 50 cycles. The tank was connected with the household water-supply. The patient's hands were dry. The right shoulder remained stiff and painful, and in the first 24 hours became much bruised. A radiograph (Fig. 125), taken five days later at a hospital, was considered to show no bone injury; a diagnosis of 'bruised arm,? hæmarthrosis' was made, and a course of radiant heat and massage was given, with some improvement in the pain. An examination under gas, forty-five days after the injury, was said to have revealed 'good and free' movement.

On Examination.—Eight weeks after the injury there was found to be no appreciable range of movement in the right shoulder, although attempts were painful. The scapular muscles were wasted; the circumference of the right arm was three-quarters of an inch less than that of the left, the right forearm a quarter-inch less than the left. No displacement of the humeral head was apparent.

X-ray Examination.—The old film taken five days after the injury was available (Fig. 125). In this projection, the overlap of humeral head and glenoid and the duplication of part of the humeral articular contour are clearly pathological; stereoscopic films were therefore taken. These appeared to show posterior dislocation of the humeral head, which was not in accordance with the clinical findings. A superior view (Fig. 126), however, showed the true state of affairs, namely an impression fracture of the humeral head opposite the glenoid.

Treatment.—Arthrodesis of the joint is under consideration.

DISCUSSION

Of the previous cases in the literature, there are five having a certain similarity: the two of Jellinek, those of Martin and of Frühmann, and the bilateral case of Simpson-Smith. In each instance the current appears to have been that of



FIG. 125.—*Case 3* Antero-posterior view. Radiograph taken five days after injury.



FIG. 126 —*Case 3*, Vertical view. Impression fracture of head of humerus.

a domestic supply or of a similar order of voltage; in each there has been a fracture of the upper end of the humerus, with or without dislocation. The fractures include fissuring (1 instance), comminution (2 instances), separation of the humeral head (2 instances), and separation of the greater tuberosity (2 instances). These five cases may be compared with the three described in the present paper.

It is clearly necessary in examples such as these to distinguish those in which mechanical violence can be eliminated and those in which the patient has fallen, so that the force of the fall may have been the sole or principal agent in causing the fracture. *Case 1* is an example of the first group, in which mechanical violence can be excluded. *Cases 2* and *3* are examples of the second, and we do not know to what extent, if any, the patient's fall may have contributed to the production of the fracture. In *Case 3* it seems unlikely that there was any relationship, because the superficial injuries from the fall occurred on the other side of the body and because we are unfamiliar with impression fractures of the humeral head as a consequence of mechanical violence.

It is interesting to speculate on the manner in which an electric shock can cause a fracture in the absence of mechanical violence. Jellinek³ showed that the passage of electric currents through bones, accidentally in man or experimentally in animals, may produce extremely delicate lines or planes of cleavage. These may be straight or zig-zag, and vary in length from a few millimetres to several centimetres. They can only just be distinguished with the naked eye, and are frequently not apparent in radiographs. Their presence was confirmed by histological examination, which failed to show any evidence of the effects of heat. Jellinek refers to the phenomenon as 'scission' or 'schisis'. The fact that bony injury of the more gross type inferred by the term 'fracture' is almost confined to the shoulder region and may be associated with crushing of part of the humeral head, or with dislocation, or with separation of the greater tuberosity, suggests that the principal agent is the uncontrolled contraction of the shoulder muscles. Whether direct fissuring of the bones by the passage of the current, as Jellinek suggests, may be present also and render them more susceptible to the muscular violence is a matter on which we have no information.

SUMMARY

1. Previously described cases of fracture resulting from electric shock are summarized.
2. Three additional cases which have been under the author's observation are described.
3. The mechanism of the direct production of fractures by electric shock is very briefly considered.

The author wishes to express his grateful thanks to Dr. Hinds Howell, who referred *Case 1* to him; to Mr. S. L. Higgs, who had charge of *Case 3*; and to Dr. Russell Reynolds, Dr. J. R. Wylie, and Dr. C. M. Roberts for the use of the radiographs in *Figs. 123, 124, and 126* respectively. He is also under an obligation to the Librarian of the Royal Society of Medicine for his assistance with the literature.

REFERENCES

- ¹ ALCOCK, N. S., "Electrical Injury of the Shoulder", *Proc. Roy. Soc. Med.*, 1935, xxviii, 1523 (Neurol. Sect.).
- ² BLENCKE, A., "Ein weiterer Beitrag zur Lunatumnekrose", *Arch. f. Orthop. u. Unfall-Chir.*, 1932, xxxi, 188.
- ³ FRÜHMANN, P., "Die Gefahr bei Behandlung von Knochenverletzungen nach elektrischen Unfällen", *Zentralb. f. Chir.*, 1931, lviii, 1815.
- ⁴ JELLINEK, S., "Roentgenologische Knochenaufnahmen bei Behandlung elektrischer Unfälle", *Wien. med. Woch.*, 1929, lxxix, 543.
- ⁵ JEX-BLAKE, A. J., "Death by Electric Currents and by Lightning", Goulstonian Lectures, No. iii, *Brit. Med. Jour.*, 1913, i, 548.
- ⁶ MARTIN, "Ueber elektrische Verbrennungen", *Zentralb. f. Chir.*, 1931, lviii, 1457.
- ⁷ PALUGYAY, J., "Knochenveränderungen bei Verletzung durch elektrischen Strom", *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1924, xxxii, 568.
- ⁸ SIMPSON-SMITH, A., "Case of Electric Shock", *Trans. Med. Soc. Lond.*, 1931, liv, 114.
- ⁹ WAGNER, W., "Lunatummalacie bei elektrischem Unfall", *Arch. f. klin. Chir.*, 1932, clxx, 483.
- ¹⁰ ZIEMKE, E., "Todesfälle durch den elektrischen Strom und ihre Beziehungen zum Unfall", *Monats. f. Unfallheilk.*, 1923, xxx, 107.

OSTEOCHONDRITIS DEFORMANS COXÆ JUVENILIS OR PERTHES' DISEASE : THE RESULTS OF TREATMENT BY TRACTION IN RECUMBENCY *

By ARTHUR L. EYRE-BROOK

OSTEOCHONDRITIS deformans coxæ juvenilis, commonly known as Perthes' disease, was first described by A. T. Legg,¹ of Baltimore, in 1909. Perthes,² after a preliminary publication in 1910, presented a classical monograph in 1913, with the result that this disease is frequently known as Perthes' disease.

ETIOLOGY

Current views upon the etiology comprise trauma, infection, constitutional affections of bone, and abnormalities of bone development.

The Traumatic Theory has been supported by an attempt to identify the epiphysal changes following reduction of a congenital dislocation of the hip with Perthes' disease, although the former do not appear to follow the well-recognized course of the latter disease, and terminate with medial flattening, in strong contrast with the marked lateral flattening of Perthes' disease. A further point, too, is that while congenital dislocation of the hip is vastly more common in girls, Perthes' disease is more common in boys. The greater frequency in boys together with some history of recent trauma have been used in support of the traumatic theory.

Trauma may of course be only of secondary importance, as in osteomyelitis. The not infrequent bilateral disease appears to make it essential to associate the traumatic theory with some predisposition, the latter possibly being of a variable nature, as suggested by Sundt. Occlusion of part of the vascular supply to the epiphysis, the result of trauma, has been supported by a number of observers on both human and experimental pathological grounds. Zemansky and Lippmann³ describe the results of section of the ligamentum teres in rabbits at various ages, and contend that in both animals and man the blood-vessels in this ligament normally gradually become occluded, and that trauma may result in their premature occlusion, with aseptic necrosis of the capital epiphysis. Lippmann⁴ describes aseptic bone necrosis in human material obtained from one of Whitman's cases.

The Infective Theory is supported by a number of distinguished observers. Platt⁵ noted otherwise unexplained pyrexia in two institutional cases, although pyrexia is rarely noted in this disease. Legg¹ and Kidner⁶ found staphylococci in one case each, and Phemister⁷ supported the infective theory from a histological study of some human material. Against this theory are advanced the facts that no abscess formation has ever been recorded and that marked X-ray signs may be seen in many apparently clinically early cases, in marked contrast to accepted infective bone conditions.

* As practised at the Royal National Orthopædic Hospital (Country Branch).

The Constitutional Affections of Bone have been enlisted to explain this disease. Rickets was blamed at one time. Durham and Outland⁸ note a diminution in both serum calcium and phosphorus, and support a constitutional disturbance. Sundt⁹ suggests an endocrinological imbalance as the underlying factor, whilst trauma, as a rule, initiates the series of changes.

Abnormalities of Bone Development may be either of the acetabulum or of the femoral head. Murk Jansen¹⁰ gives an ingenious explanation wherein the condition of ischium vara or of an abnormally shallow acetabulum results in point contact only between the acetabulum and the femoral head. The resultant excessive local pressure in somewhat softened bone results in Perthes' disease and deformity of the femoral head, mainly of an adaptive nature. A survey of a number of radiographs with a view to discovering any degree of ischium vara convinced me that without accurately centred X rays this could not be determined.

Delitalia¹¹ considered that the abnormality of bone growth was in the femoral metaphysis. He pictured Perthes' disease as similar in pathology to a slipped femoral epiphysis, save that the abnormality was transferred from the medial to the lateral region of the metaphysis.

CLINICAL PICTURE

The clinical material upon which this paper is built comprises 41 cases, varying in age of onset from 3 years and 3 months to 16 years, and showing a marked preference for the period 4-9 years, in which semi-decade lay 72 per cent of the cases. The sex incidence is male/female as 31/10; that is, the disease is three times as common in boys as in girls. Only four cases of bilateral disease were encountered.

In considering the clinical picture, those cases seen in the acute stages showed spasm, which appeared considerably in excess of the pain complained of, this always being slight. The prominence of the head in Scarpa's triangle and of the great trochanter were constantly observed, and, even after quiescence of the disease had been reached, there was usually sufficient to determine clinically the side involved. All other signs rapidly disappeared once treatment was begun.

X-ray Signs of Perthes' Disease.—These are divisible into the early signs (*Figs. 127-130*), unfortunately only too rarely seen, and the better-known typical sequence of the later stages (*Figs. 131-135*).

Early Signs.—There are five of these: (1) Increased density of the epiphysis; (2) Increased depth and clarity of the joint space; (3) Early flattening of the epiphysis; (4) Early metaphysial 'cavitation'; (5) Courtney Cages's sign.

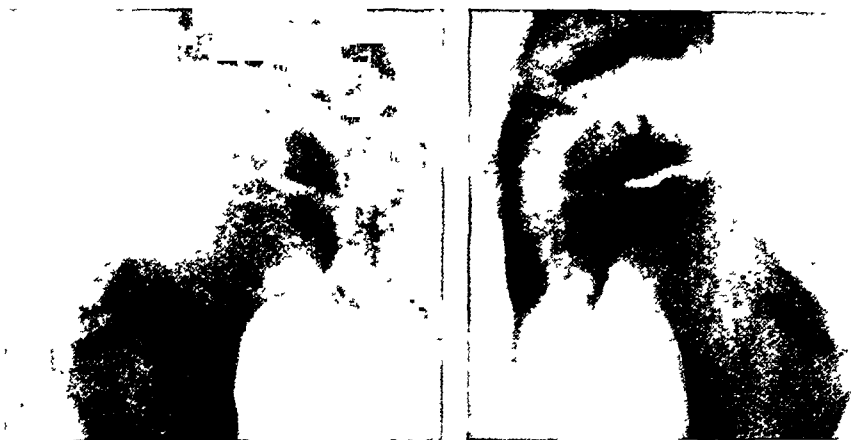
Increased density of the epiphysis appears to precede flattening, and in the earliest radiographs is only accompanied by the increase in depth and clarity of the joint space, which latter as a rule is quite a noticeable feature. These joint-space signs have been noted by a number of observers, particularly of the French school, and are presumably the result of an effusion into the joint, although the adduction of the hip does not lend support to this suggestion.

Early flattening of the epiphysis rapidly follows the above, and soon becomes one of the most striking of the X-ray signs of the disease.

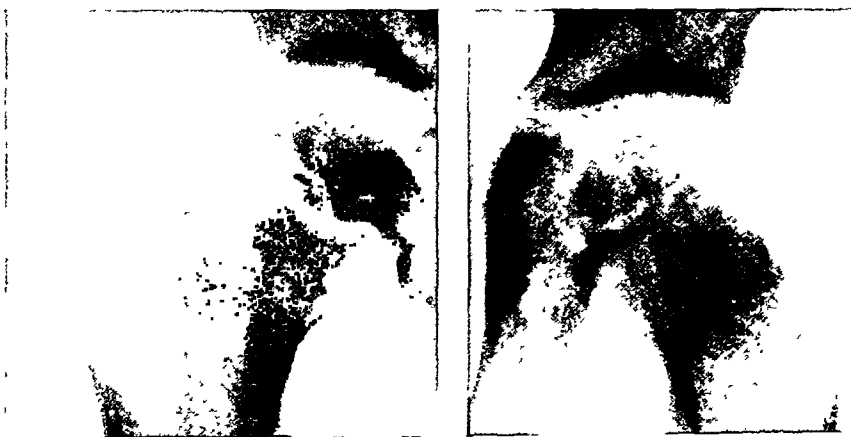
Early localized metaphysial rarefaction or 'cavitation' reminds us that this disease is not confined to the epiphysis in its manifestations. A small translucent

area in the metaphysis may be the most evident of the X-ray signs in an early radiograph, as occurred in one of our cases.

Courtney Cages's¹² sign is the lateral metaphyseal erosion that develops on the affected side and contrasts with the normal straight line of junction of the metaphysis with the epiphysis. This sign was definitely present in three out of six very early cases, but does not appear to be constant or of very great assistance in early diagnosis.



FIGS. 127, 123.—Case 15. A. W. Aged 8 years. Perthes' disease of the right hip, showing increased depth and clarity of the joint space, increased density of the epiphysis without collapse, and Courtney Cages' sign.



FIGS. 129, 130.—Case 3. D. R. Aged 4 years. Perthes' disease of the right hip, showing increased density of the epiphysis without collapse, increased depth and clarity of the joint space, metaphyseal 'cavitation', and a thickening of the femoral neck from subperiosteal laying-down of bone.

Later Signs.—These, the better-known signs of Perthes' disease, are (6) Flattening, fragmentation, and further condensation of the epiphysis; (7) Broadening of the neck of the femur; (8) Confluent 'cavitation' of the metaphysis; (9) Partial collapse of the 'cavited' metaphysis; (10) Regeneration of the more or less deformed epiphysis; (11) Condensation of the regenerated epiphysis;

- (12) Sometimes partial disappearance of the epiphysial line in its outer portion;
 (13) The appearance of the transverse cervical line; (14) Adaptive acetabular changes



FIG 131—Case 21 W E Aged 10 years. Much metaphysal 'cavitation'. The prognosis is bad because of the danger of severe metaphysal collapse.



FIG 132—Case 18 A E Aged 7 years. Showing the formation of a marked beak, resulting in severe metaphysal deformity.



FIG 133—Case 16 B D Aged 9 years. Illustrating the thickening of the neck of the femur by laying-down of subperiosteal laminae of bone along the upper surface of the neck.



FIG 134—W M Aged 9 years. Showing the transverse cervical line and the partial disappearance of the epiphysial line.

The broadening of the neck results from two processes—the laying-down of laminae of subperiosteal bone along the superior surface of the neck, and the downward

protrusion of the inner portion of the metaphysis, this latter being known as the beak.

Severe metaphysial 'cavitation' is a serious prognostic factor, as it is liable to result in considerable metaphysial collapse and deformity, particularly of the outer portions.

Regeneration of the epiphysis is very interesting to watch as it gradually takes visible form on the X-ray films. The outline creeps on to the upper surface of the neck, even reaching and fusing with that of the great trochanter. Any protrusion of the head beyond the acetabular margin must be taken as deformity, even when the epiphysis is agreeably round.

While the epiphysis is usually involved *in toto*, there is always a much greater degree of pathological change to the outer side. Such mushroom types as were

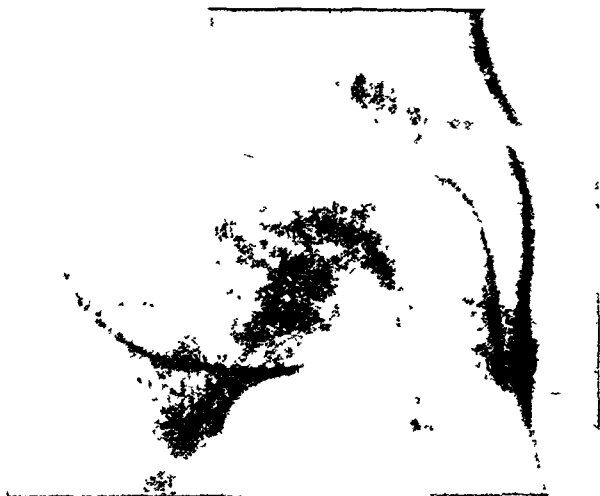


FIG. 135 —P. B. Aged 11 years. Like Fig. 134, this shows the transverse cervical line and the partial loss of the epiphysial line.

present in this series occurred as the late deformed result of former cap types which had received treatment deficient either in intensity or duration.

Partial disappearance of the epiphysial line only occurred in the more severely deformed cases, and is illustrated in one of the radiographs shown.

The transverse cervical line crosses the neck at various levels, according to the degree of deformity present, and is quite characteristic of these severer cases. It is presumably the everted edge of the metaphysis, and always starts in the beak. From the greater encroachment on internal rotation, I conclude that it is on the anterior surface of the neck.

The acetabulum shows adaptive changes after the disease has been in progress for some time. No early primary changes were noted.

TREATMENT AND PROGNOSIS

In turning to the question of treatment, we note that a great diversity of opinion has existed. Legg¹³ in 1927 summed up the views of many surgeons with the remark that, "while a process suggesting weakness of bone structure is going

on, it is theoretically sound to allow no weight-bearing; but in practice, relief from weight-bearing in no way affects the end-results." I think at the present day most surgeons would at least agree with Sundt,¹⁴ who stated that "treatment directed to the elimination of weight-bearing has no proved influence on the train of morbid changes, but its application is indicated during the stage of prominent symptoms."

The reason for the prevalence of such pessimistic views on this question is undoubtedly the advanced stage of the disease at which treatment is so often begun. The disease is so insidious that treatment is often aimed at preventing that which has already occurred. The results possible with efficient treatment of early cases, as shown in this article, make it clear that while complete freedom from all stresses "has no proved influence on the train of morbid changes", it has a very considerable influence on the results of these morbid changes.

The aim of treatment should be: (1) Clinically, to maintain a full range of movements with no predisposition to osteo-arthritis from a misfit in the acetabulum. (2) Radiologically, to produce a round head, adapted to the acetabulum, approximating as closely as possible to the normal. The radiological aim, when attained, will certainly result in clinical perfection in function of a permanent nature, but a good early clinical result may be present in a case showing considerable deformity and which is very prone to crippling arthritis.

The prognosis in this disease has been stated to depend upon: (1) The type of head—cap or mushroom; (2) The age of the patient. Most observers will not deny the influence of the following two factors also: (3) The duration of the disease before treatment is instigated; (4) The efficiency of the treatment.

I have not been able to endorse the prognostic value of the type of head as laid down by Legg¹³ in 1927. That the cap type takes longer to cure than the mushroom, as he stated in 1929,¹⁵ one must accept. It is then well on its way to consolidation, but the final result is always bad.

My series amply confirms the great importance of age in relation to prognosis, as laid down by Flemming-Møller.¹⁶ There appear to be two factors to consider. The most important seems to be the natural course of the disease at different ages, but there is a second easily recognized factor, which is the purely mechanical one of the amount of weight carried, of the crushing forces acting on the diseased femoral head. Both these factors alter for the worse with advancing years.

The third prognostic factor is the condition of the epiphysis and metaphysis when treatment is begun. It is remarkable the degree of deformity that has often occurred by the time the child is first brought to the doctor. The head may be calcified and clearly seen, or in part decalcified and in part represented by dense calcified masses. In these latter we are still able to recognize the degree of deformity. The distance between the epiphysial line and the roof of the acetabulum gives a clear conception of the amount of collapse that has occurred in the softened head, of which the radiograph may give no outline at all. From a study of the serial radiographs of many cases, it appears to me that the flattening of these heads is permanent, and can only be relieved by some increase in depth of the epiphysis from natural growth. There does not appear to be any natural resilience or recoil to benefit by extension, nor does lateral capsular tension restore the shape.

And so we pass to the fourth point in prognosis, which is the efficiency of treatment. Treatment must be concentrated upon the avoidance of further deformity while the disease runs its course of softening, local condensation of calcium

content, fragmentation, recalcification, and consolidation. I picture each head as being in a softened condition and suffering progressive deformity from stresses, which are greater with increasing years, and I look upon the best treatment as that which will completely rid the femoral head of all stresses and allow of uninterrupted consolidation and natural growth. The treatment must relieve the head of all crushing forces, not only those of weight-bearing but also those of muscular contraction.

One other point to be considered is whether we are to encourage immobilization or movement of the affected leg. The former leads to a certain amount of wasting of muscles and stiffening of joints. The latter, while avoiding these complications, may also be of value in the treatment of the later cases by promoting a roundness of the head without any further flattening. It probably rounds the sharp edges of the head, while efficient extension preserves the maximum height in the more central portions.

With these points in mind, let us consider the methods of treatment in general use to-day. They are divisible into ambulatory and recumbent. Of the former, we have: (1) The weight-relieving calliper; (2) The patten-ended calliper; (3) Crutches, patten, and calliper.

The simple calliper merely reduces weight-bearing, but the patten-ended calliper is efficient in the abolition of all weight-bearing in the protected hip. The complete freedom from weight-bearing, however, does not protect the femoral head from all crushing forces. Trumble¹⁷ has shown how the abductors of the hip crush the head in holding the pelvis horizontal, while moving the other leg forward. Trumble's splint therefore takes the weight centrally and dispenses with the aid of the abductors in walking, thus relieving the femoral head of all crushing forces. The third ambulatory method of treatment in common use is the calliper on the diseased side, patten on the sound side, and crutches. This fulfils all our requirements. When this system of treatment is carefully executed, the calliper and patten being worn continuously, there can be little objection to the treatment. But can this often be obtained? It will be noticed that over 72 per cent of the cases in the present series were under the age of 9 years. Can children of this age be expected to carry out this treatment in its entirety? The older children may be satisfactorily treated by this method, but in the younger children the treatment must fall very short of its theoretical possibilities.

Now let us turn to recumbent treatment and consider the methods available. These are: (1) A plaster spica; (2) Robert Jones's frame; and (3) Simple sliding traction in bed. The last of these three, and incidentally by far the simplest and cheapest, appears to meet all our requirements. The plaster spica involves much undesirable wasting and stiffening of joints, as does the Jones's frame, although to a lesser degree.

TREATMENT BY TRACTION IN RECUMBENCY

At the Royal National Orthopædic Hospital the method employed consists of sliding traction with a 6 to 10 lb. weight and pulley, the foot of the bed being raised, and no splinting being used on the affected leg. Alternately Pugh's extension is perfectly satisfactory. With either method we obtain with a fair certainty our aim of reasonable freedom of movements with the avoidance of all crushing forces

acting upon the diseased femoral head. The progress is controlled by radiographs taken every 3 to 4 months, and treatment ceases when calcification and consolidation are complete. Our average duration of treatment is 18 to 24 months, but a longer period is needed in a number of cases. This treatment is such that we are confident that no known non-operative procedure can give these femoral heads a better chance of recovery. But are we obtaining this end with a minimum of expenditure and least possible interference with the normal life of the child? I believe that this is so in most cases. The children are too small to fulfil to the letter the full requirements of efficient ambulatory treatment. Education is carried out without interruption in one of our orthopaedic hospitals, and the general health will certainly benefit. The need for removing the child from its home is to be regretted in most cases.

Results.—Our cases are divided into two series, those treated by extension and those otherwise treated. The former were all treated at the Royal National Orthopaedic Hospital (Country Branch), where this method of treatment has been in use since 1930. In considering results, I have grouped the cases into those under 7 years and those over 7 years of age, the former to be known as *Group I* and the latter as *Group II*. Results are so dependent on the condition of the head at the inception of treatment that I have divided the original radiographs into three stages. A Stage I case presents an original radiograph showing a femoral epiphysis with little or no flattening—in fact the diagnosis may require confirmation by later radiographs. These cases can be induced to give results little short of the normal. Stage II cases show typical flattening, fragmentation, and cervical thickening. Stage III cases show more advanced changes, with recalcification.

Epiphysial Index.—For statistical purposes, I have introduced a quotient to indicate the result of treatment—

$$\frac{\text{Height of epiphysis}}{\text{Breadth of epiphysis}} \times 100 = \text{Epiphysial Index}$$

While admitting that Perthes' disease is not solely an epiphysial lesion, the metaphysial changes are nearly always proportional to the epiphysial deformity; hence the index is a fair representation of results. The normal varies somewhat with age and also from case to case. Under 7 years of age it varies from 45 to 55, while over 7 years of age it has a range from 35 to 45. It will be noted from *Table I* that with Stage I cases in *Group I*, i.e., under 7 years of age, we can approach near to the ideal of a normal head. Some slight interference with the even curve of the head is the only hint of healed Perthes' disease in some of the best results of traction treatment. The control figures do not boast of many Stage I cases in the younger group, but if we turn to Stage II cases we note an average 10 per cent improvement in the epiphysial indices in the cases treated by traction in recumbency. These control cases have been carefully treated, and many have been confined to bed for periods of six to eight months, yet there is a 10 per cent average difference in the epiphysial indices of the two series. The difference in results cannot be attributed to lack of specialization, but must be related to the treatment, the details of which are given in the table. In these younger cases the results appear amply to repay the additional trouble taken. The results are definitely superior to those obtained by other methods, and some of the very early cases give results that it would be difficult to excel.

Table 1.—RESULTS OF VARIOUS METHODS OF TREATMENT IN PERTHES' DISEASE IN TERMS OF THE EPIPHYSIAL INDEX

CASE No.	INITIALS	AGE AT ONSET	TREATMENT	EPHYSICAL INDEX	TREATMENT	AGE AT ONSET	INITIALS	CASE No.
1	L. H.	4	Extension	30 months	Average 39 Early Stage I	26	R. A.	23
2	J. C.	4½	"	19 "	{ 48 } { 36 } { 28 } { 48 } { 40 }			
3	D. R.	4	"	14*				
4	O. S.	6	"	17				
5	M. B.	6	"	18				
6	D. H.	3½	"	30	Normal 45/55			
7	D. M.	5	"	16	Average 27 Medium Stage II	{ 30 } { 23 } { 20 }	J. E. K. M. J. W.	24 25 26
8	T. B.	5	"	20				
9	A. M.	5½	"	15				
10	H. H.	6	"	17				
11	K. W.	5	"	21			W. W.	27
12	L. C.	5	"	12			A. D.	28
13	P. B.	6	"	19			L. H.	29
14	W. M.	6	"	6			W. W.	30
15	A. W.	7	"	18				
16	B. D.	9	"	18				
17	C. C.	11	"	13				
18	A. E.	7	"	19	Average 23 Early Stage I	21	A. G.	32
19	F. B.	7	"	15			A. B.	33
20	P. A.	9½	"	16				
21	W. E.	10	"	15			R. M.	34
22	A. I.	11	"	22			L. R.	35
			"	12			R. S.	36
			"	12			V. H.	37
			"	12			G. C.	38
			"	12			P. B.	39
			"	12			E. A.	40
			"	12			B. D.	41



FIG 136—Case 4 O.S. Aged 5 years Group I, Stage I case Treatment Extension for 17 months, no calliper Radiograph showing appearance when treatment was begun (Note early signs of the disease)



FIG 137—Case 4 After 5 months' treatment by extension



FIG 138—Case 4 After 13 months' treatment



FIG 139—Case 4 Appearance 12 months after discharge Result Epiphysial Index, 48

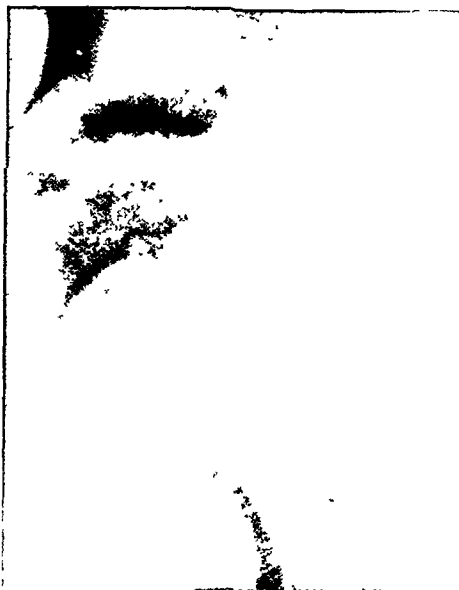


FIG. 140. *Case 16. B.D. Aged 9 years. Group II, Stage I case. Treatment: Extension in recumbency for 18 months. Radiograph at onset of treatment*



FIG. 141.—*Case 16. After 9 months' treatment*

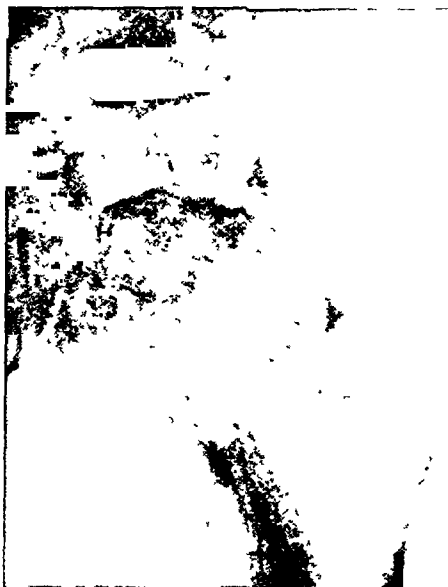
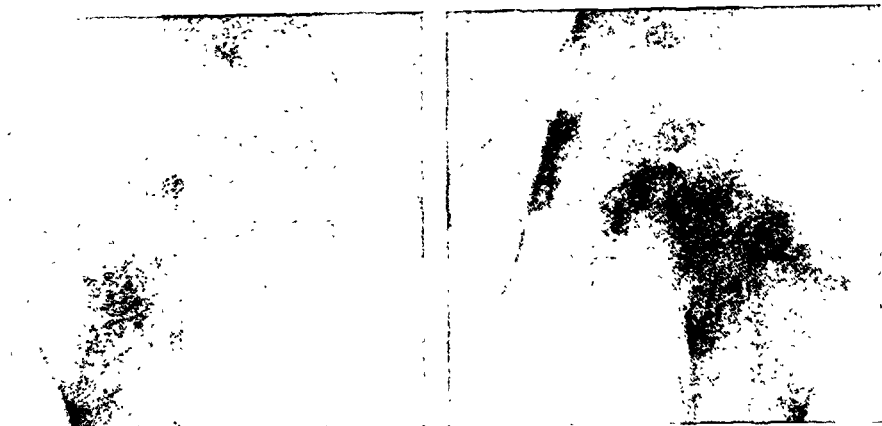


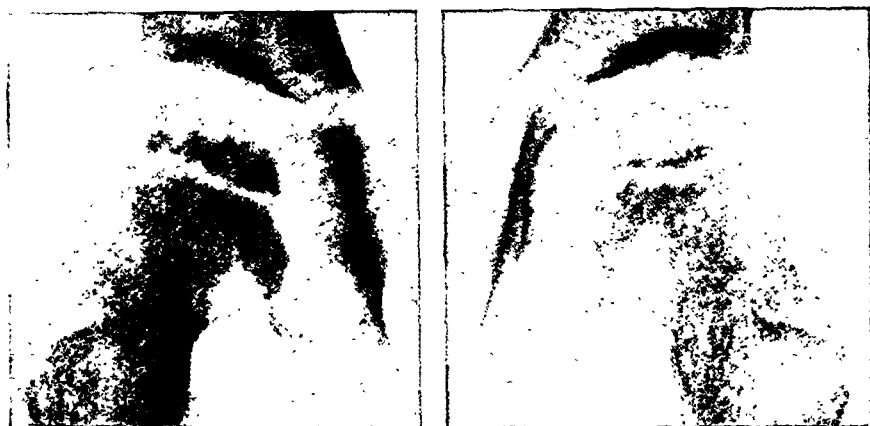
FIG. 142.—*Case 16. After 15 months' treatment.*



FIG 143.—*Case 16. After 4 years had elapsed since treatment was commenced. Result: Epiphysial Index, 20*



FIGS. 144, 145.—Case 2. J. C. Aged 41 years. Group I, Stage I case of bilateral disease. Treatment: Extension in recumbency for 19 months. Radiographs before treatment was commenced.



FIGS. 146, 147.—Case 2. Radiographic appearance at conclusion of treatment. Result: Epiphysial Indices, R. 36, L. 48.

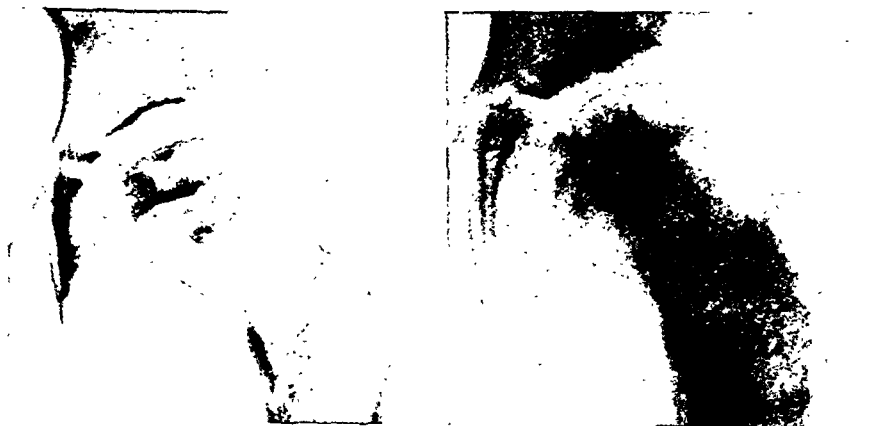


FIG. 148.—Case 5. M. B. Aged 6 years. Group I, Stage I case. Treatment: Extension for 18 months; no calliper. Radiographs before treatment was commenced.

FIG. 149.—Radiograph 12 months after treatment was completed. Result: Epiphysial Index, 40.



FIG. 150.—Case 9. A. M. Aged 5½ years. *Group I, Stage II case. Treatment:* Extension for 13 months and 2 months free in bed; no calliper. Radiograph taken at beginning of treatment.
FIG. 151.—Appearance 2½ years later. *Result:* Epiphysial Index, 20.



FIG. 152.—Case 10 H. H. Aged 6 years. *Group I, Stage II case. Treatment:* Extension for 16 months, free in bed for 1 month; no calliper. Radiograph taken when treatment was started.
FIG. 153.—Showing X-ray appearance 2½ years later. Epiphysial Index, 30.

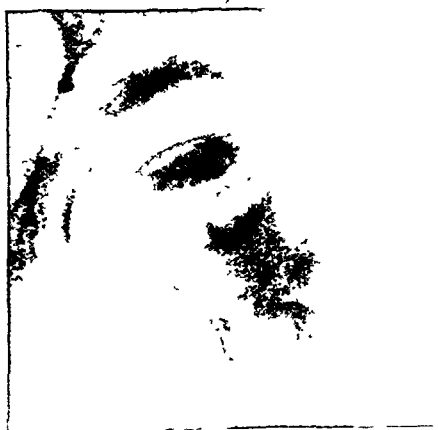


FIG. 154.—Case 11. K. W. Aged 5 years. *Group I, Stage II case. Treatment:* Extension for 17 months, free in bed for 4 months; no calliper. Radiograph taken when treatment was commenced.
FIG. 155.—Radiograph taken 3½ years later. Epiphysial Index, 20; well-adapted head.



FIG. 156.—Case 7. D. M. Aged 5 years. Group I, Stage II case. Treatment: Extension for 15 months, no calliper. Shows X-ray appearance at start of treatment.

FIG. 157.—Radiograph taken 2 years 9 months later. Epiphysial Index, 30.



FIG. 158.—Case 6. D. H. Aged 3½ years. Group I, Stage II case (late). Treatment: Extension for 20 months, free in bed 10 months; no weight-bearing for a total of 30 months. X-ray photograph before treatment was started.

FIG. 159.—Radiograph at completion of treatment. Epiphysial Index, 34; head not a good shape.



FIGS. 160, 161.—Case 8. T. B. Aged 5 years. Group I, Stage II case. Treatment: Extension for 20 months. Result: Epiphysial Index, 30. Radiographs illustrate severe metaphysial 'cavitation', with resultant severe metaphysial deformity from collapse.



FIGS. 162, 163—K. M. Aged 4½ years. *Group I, Stage II case* Treatment None, refused. *Result:* Deformed head with an Epiphysial Index of 23. The child would have had a far more deformed head had he been older, thus emphasizing the importance of the age factor. Interval between X-ray photographs, 3½ years



FIG. 164—P. B. Aged 10 years. This is the original radiograph of the case of a boy who had received no attention for pain in the hip and a lump, these symptoms having been present for the last 18 months. Recumbent treatment will not be repaid in any such case. Again we see the importance of age and the insidious nature of this disease.

What of the older, *Group II*, cases? Here the results are not so encouraging. In Stage I cases it appears that the results are better than those of the control series, and the index in one case actually reached within normal limits. The difference is not so marked, however, and the later cases seem to respond equally well to ambulatory methods.

From a statistical survey it appears that the results in all cases under 7 years of age and in those cases over this age in a very early stage of the disease justify the extra trouble and expense of the treatment by traction in recumbency. The

remaining cases, and in particular the late ones, certainly do not respond adequately to this treatment.

The illustrations *Figs. 136-164* are mainly confined to some of our better results, obtained in *Group I* cases in Stages I and II of the disease. These not only illustrate the indices obtained but also the roundness of the heads, well adapted to the normal acetabulum, and thus obviating the risk of any later arthritis. Included in the illustrations are two interesting untreated cases, which demonstrate admirably the paramount importance of age in prognosis (*Figs. 162-164*). Both cases gave poor results, but the older case is far more deformed although the disease must have been in progress for a longer time in the younger case.

CONCLUSIONS

1. In considering the etiology, whether the traumatic or infective theory is favoured, it appears necessary to stipulate some predisposing factor, either constitutional affection of bone or abnormality of bone development, to account for the frequency of bilateral disease.

2. Prognosis in Perthes' disease is dependent upon: (a) The age of the child, which controls both the severity of the natural course of the disease and the stresses to which the diseased femoral head is to be subjected; (b) The stage to which the disease has progressed when treatment is begun; (c) The effectiveness of treatment.

3. Efficient ambulatory methods of treatment are the calliper, crutches, and patten, and the Trumble's splint. Both these are only really suitable for children over 7 years of age, and each requires constant and understanding supervision in younger children. Traction in recumbency provides the really satisfactory treatment for children under 7 years of age or over this age in a really early stage of the disease. Two methods of providing this treatment are recommended: (a) Sliding traction with weight and pulley; and (b) Pugh's extension. Results, as shown by the illustrations (*Figs. 136-164*) and *Table I*, are provided to support this contention.

4. The results are permanent, as is shown by following up some of the better results for a number of years.

5. We have never obtained a normal head, but we can approach near to this ideal.

6. Finally, I would stress the fact that the clinical results are grossly complimentary, quite severely deformed heads giving movements and function little short of the normal. We must turn to the X-ray picture and the later complications for a truer perspective. The deformed heads are all candidates for early crippling arthritis. We must not allow our 'cures' to be this fertile soil for arthritis. We must aim at permanent and anatomical and not merely temporary functional successes.

In conclusion, I wish to express my gratitude to all those surgeons in London and Bristol who gave me free access to their cases of Perthes' disease, to Mr. C. F. Coombs for helping me with the photography, and, above all, to Mr. H. J. Seddon for his help and advice in many matters.

REFERENCES

- ¹ LEGG, *Boston Med. and Surg. Jour.*, 1910, clxii, 202.
- ² PERTHES, *Arch. f. klin. Chir.*, 1913, ci, 779.
- ³ ZEMANSKY and LIPPMANN, *Surg. Gynecol. and Obst.*, 1929, xliii, 461.
- ⁴ LIPPMANN, *Amer. Jour. Surg.*, 1929, vi, 785.
- ⁵ PLATT, *Brit. Jour. Surg.*, 1922, Jan., 366.
- ⁶ KIDNER, *Amer. Jour. Orthop. Surg.*, 1916, xiv, 339.
- ⁷ PHEMISTER, *Arch. of Surg.*, 1921, March, 221.
- ⁸ DURHAM and OUTLAND, *Jour. Bone and Joint Surg.*, 1928, x, 301.
- ⁹ SUNDT, *Lancet*, 1921, i, 1065.
- ¹⁰ MURK JANSEN, *Jour. Bone and Joint Surg.*, 1923, xxi, 265.
- ¹¹ DELITALIA, *Amer. Jour. Orthop. Surg.*, 1915, xii, 555.
- ¹² COURTNEY CAGE, *Brit. Jour. Radiol.*, 1933, vi, 295.
- ¹² LEGG, *Jour. Bone and Joint Surg.*, 1927, ix, 26.
- ¹⁴ SUNDT, *Zentralb. f. Chir.*, 1920, May, 29.
- ¹⁵ LEGG, *Amer. Jour. Surg.*, 1929, vi, 793.
- ¹⁶ FLEMMING-MØLLER, P., *Acta Radiol.*, v, 1-36 —.
- ¹⁷ TRUMBLE, *Brit. Med. Jour.*, 1935, May 25, 1070.

Literature not specifically referred to :—

- LEGG, *Surg. Gynecol. and Obst.*, 1916, March, 307.
 PATTISON, *Brit. Jour. Surg.*, 1928, xvi, 89.
 WHITMAN, *Amer. Jour. Surg.*, 1928, iv, 185.
 WHITMAN, *Ibid.*, 1929, vi, 791.
 ZEMANSKY, *Ibid.*, 1928, iv, 169.
 ROCHER, *Arch. franco-belges de Chir.*, 1927, xxx, 24.
 LANG, *Jour. Bone and Joint Surg.*, 1932, xiv, 563.
 BOZAN, *Ibid.*, 885.
 SNODGRASS, *Ibid.*, 314.
 CAMITZ, *Acta chir. Scand.*, 1934, lxxviii, 521.
 MILNER and HU, *Arch. of Surg.*, 1933, xxvii, 645.
 LEWIS, *Radiology*, 1934, xxii, 188.
 CALDWELL, *South. Med. Jour.*, 1934, xxvii, 402.
 WALDENSTROM, *Acta orthop. Scand.*, 1934, v, 1.
 BOZSAN, *Jour. Bone and Joint Surg.*, 1934, xvi, 75.
 DANFORD, *Ibid.*, 516.
 BRAILSFORD, *Lancet*, 1935, i, 1487.
 KING, *Localized Rarefying Conditions of Bone*, 1935.

EXPERIMENTAL SURGERY

OBSERVATIONS ON THE EXPERIMENTAL PRODUCTION OF PEPTIC ULCER IN THE ILEUM

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PEPTIC ulcer in the ileum is rare. It is practically always associated with the presence of a Meckel's diverticulum which contains heterotopic gastric mucosa in its wall.^{1, 3, 6} Recent reviews of the literature by Johnson and Renner⁵ and Schullinger and Stout¹² revealed the fact that there had been reported not more than fifty cases of peptic ulcer of the Meckel's diverticulum in which gastric mucosa

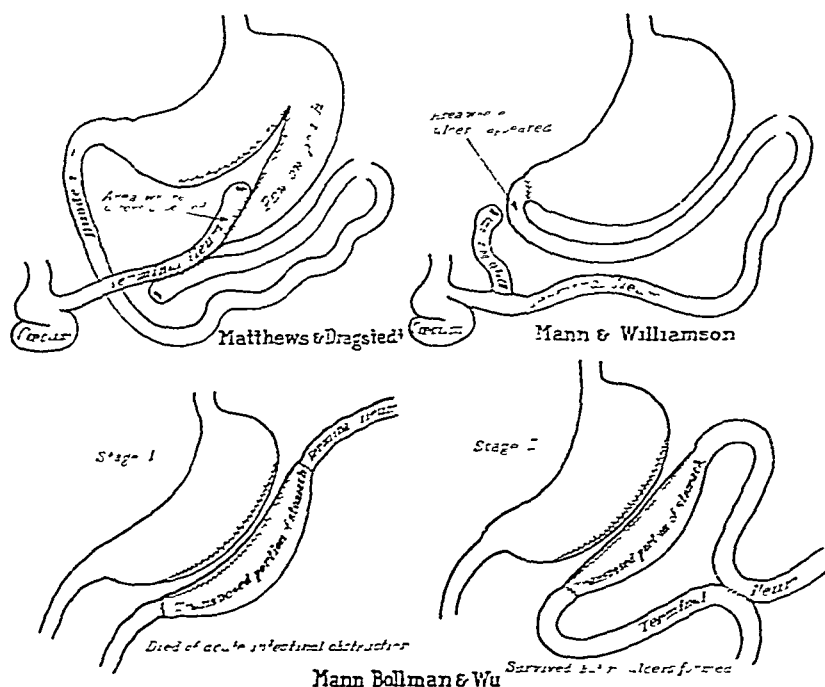


FIG. 165—Various procedures in diagrammatic form for the experimental production of peptic ulcer.

was identified on histologic examination. As the aberrant gastric mucosa must have been present from the time of birth, it may be assumed that the development of the ulcerative process is due to the operation of certain superimposed factors. The object of this study was to reproduce the lesion experimentally and determine some of the factors concerned.

Matthews and Dragstedt⁹ produced an experimental counterpart of the disease by making a small Pawlow pouch emptying into an isolated loop of the lower ileum (*Fig. 165*). In all of their six dogs large chronic ulcers were found in the intestinal mucosa adjacent to the gastric pouch between thirty-four and ninety-one days after operation.

Mann, Bollman, and Wu⁷ attempted to reproduce this condition by carrying out a series of experiments on dogs in which the fundic portion of the stomach was resected and converted by partial closure into a tube. This tube formed by part of the stomach was then interpolated between the cut ends of the lower ileum by end-to-end anastomoses (*Fig. 165*). All the animals invariably died from acute obstruction, with the blockage of the intestinal contents occurring at the site of the gastro-intestinal anastomoses. Consequently, they modified the procedure by adding an entero-enterostomy between the loops of ileum immediately proximal

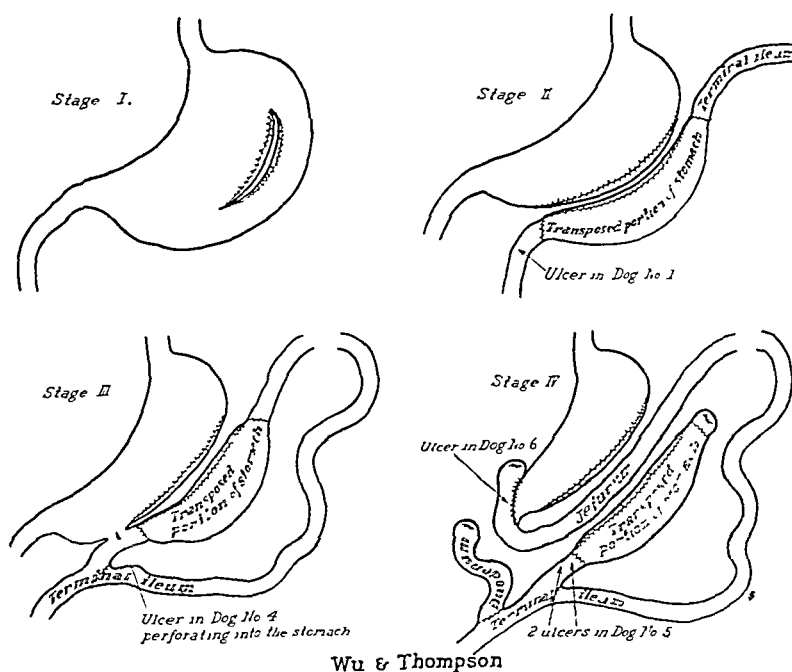


FIG. 166 —Diagrams showing the authors' procedures for the experimental production of peptic ulcer.

and distal to the transposed stomach. All the animals in this series survived, but none of them had developed an ulcer in the gastro-intestinal tract at the end of a year.

The present report is based upon the results obtained on seven of the dogs used for this study. Twenty-three operations, including three different procedures and exploratory laparotomies, were performed under inhalation ether anaesthesia and with an aseptic technique. All the anastomoses and closures of the stomach and the intestines were effected by the use of plain No. 1 catgut for the mucosa and fine silk for the seromuscular layer (*Fig. 166*).

The first two stages were really parts of a single procedure. For the first stage, the stomach along the greater curvature from the fundus to the pyloric

antrum was converted into a tube with the lumen of the latter communicating with that of the stomach proper through the two ends. After an interval of two or three weeks, the second stage was carried out, when the tube constructed from the stomach was resected and interposed iso-peristaltically between the divided ends of the ileum about 50 to 60 cm. proximal to the ileocaecal junction by end-to-end anastomoses.

It was found that by executing these procedures in stages intestinal obstruction did not occur. This is attributed to the acquisition of some sort of motor function in the tube of stomach during the interim between operations. However, fluoroscopic examination after the administration of a barium mixture failed to demonstrate any peristalsis.

Only two of the seven dogs developed an ulcer in the ileum. The first dog died from generalized peritonitis on the second day following operation. Necropsy revealed an acute perforated ulcer, measuring 3×1.5 cm. across, on the anti-mesenteric side of the ileum immediately distal to the transposed stomach. The second dog, dying also from generalized peritonitis on the eighth post-operative day, showed an acute perforated ulcer, about 5 mm. in diameter, on the anti-mesenteric side of the ileum just proximal to the interpolated tube of stomach.

Ulcerative lesions could not be found in the remaining five dogs. One of them was killed by an accident on the fiftieth day following operation, while the other four were subjected to an exploratory laparotomy 15, 28, 35, and 55 days after the interpolation of the tube of stomach in the ileum.

In order to determine the role played by the alkaline intestinal contents in preventing the development of a peptic ulcer, a further procedure (Stage III) was carried out on each of the four dogs that survived. The ileum just above the transposed stomach was divided. The distal end was closed, while the proximal end was anastomosed to the side of the ileum about 5 to 10 cm. below the tube of stomach. It is to be noted that the experimental set-up at this time simulates in principle that of Matthews and Dragstedt.

One of the four dogs was found to have developed a chronic peptic ulcer at an exploratory coeliotomy twenty days after side-tracking the intestinal stream. The ulcer was located in the ileum just distal to the transposed stomach. It measured about 1 cm. in diameter. Its margins were indurated and its base had perforated into the wall of the stomach proper, to which it was firmly adherent (*Fig. 167*).

Exploratory laparotomies on the other three dogs 20, 28, and 34 days following the deviation of the alkaline intestinal contents from the vicinity of the transposed stomach yielded negative results.

What is the effect in the vicinity of the transposed stomach when conditions leading to peptic ulcer formation are established elsewhere? To answer this question, the classical Mann and Williamson's procedure for the production of peptic ulcer was done on the three dogs left in the series (*Fig. 165*). This consisted in replacing the duodenum by the jejunum and draining the alkaline duodenal contents into the lower ileum (Stage IV).

One of the dogs died from generalized peritonitis eight days subsequent to the last procedure. Necropsy showed two ulcers in the ileum immediately distal to the transposed stomach, but no lesion in the jejunum which was anastomosed to the stomach proper. One of them, measuring 1×4 cm. across and located on the anti-mesenteric side of the bowel, was perforated. The other one was

located opposite the first one and measured about 1 cm. in diameter. Another dog died on the twenty-eighth post-operative day from generalized peritonitis arising from a perforated chronic ulcer measuring about 1 cm. in diameter and located on the superior wall of the jejunum directly beyond the gastrojejunal anastomosis. The ileum on either side of the transposed stomach showed only irregular areas of superficial ulceration. An exploratory laparotomy performed on the third dog fourteen days after the establishment of surgical duodenal drainage showed no lesion in either the jejunum or the ileum.

The results of this investigation seems to suggest several things. The importance of acid as a factor in the development of peptic ulcer has been demonstrated by the experimental studies of Exalto,² Mann and Williamson,⁸ Matthews and Dragstedt,⁹ Morton,¹¹ McCann,¹⁰ Wu,¹³ and many others. On the other hand, the presence of heterotopic gastric tissue in the ileum only occasionally, but not invariably, leads to the development of peptic ulcer. While the difference in the outcome may depend upon a qualitative or quantitative variation of the aberrant

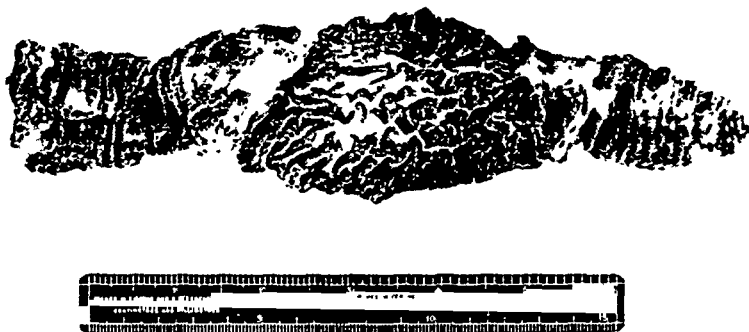


FIG. 167.—Photograph of specimen obtained at an exploratory laparotomy twenty days after deviating the intestinal current from the vicinity of the transposed stomach. The gastric tissue is in the middle, the proximal closed end of ileum on the right, and a chronic ulcer in the distal ileum on the left.

gastric secretion or of the ileac contents, the theory of diathesis or individual susceptibility to the disease may also be invoked. That the alkaline ileac contents exert a protective or neutralizing influence against the acid gastric juice is shown by the development of peptic ulcer following the deviation of the intestinal current from the vicinity of the transposed stomach. The fact that ulcers are not invariably produced by such procedures calls for no explanation so long as there exists the possibility of regurgitation of intestinal contents into the side-tracked portion. Finally the development of ulceration in the ileum after the operation of surgical duodenal drainage to produce a jejunal ulcer had been added to the other two procedures is somewhat suggestive of a reflex nervous or hormonal factor. Until secretory nerves to the islands of gastric tissue in a Meckel's diverticulum have been demonstrated, a hormonal mechanism like that established by Ivy and Farrell⁴ in autogenous pouches of the fundic portion of the stomach transplanted subcutaneously in dogs appears to be more important from the clinical point of view.

In conclusion, it may be stated that peptic ulcer in the ileum has been produced experimentally by one or a combination of several procedures. The observations made in this study seem to lend support to the hypothesis that the development of peptic ulcer in a Meckel's diverticulum containing heterotopic gastric mucosa depends on biologic, chemical, and possibly reflex nervous or hormonal factors.

REFERENCES

- ¹ ASCHNER, P. W., and KARELITZ, S., *Ann. of Surg.*, 1930, xci, 573.
- ² EXALTO, J., *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, xxiii, 13.
- ³ GREENWALD, H. M., and STEINER, M., *Amer. Jour. Dis. Child.*, 1931, xlii, 1176.
- ⁴ IVY, A. C., and FARRELL, J. I., *Amer. Jour. Physiol.*, 1925, lxxiv, 639.
- ⁵ JOHNSON, L. B., and RENNER, G., jun., *Surg. Gynecol. and Obst.*, 1934, lix, 198.
- ⁶ LINDAU, A., and WULFF, H., *Ibid.*, 1931, liii, 621.
- ⁷ MANN, F. C., BOLLMAN, J. C., and WU, P. P. T. Unpublished data.
- ⁸ MANN, F. C., and WILLIAMSON, C. S., *Ann. of Surg.*, 1923, lxxvii, 409.
- ⁹ MATTHEWS, W. B., and DRAGSTEDT, L. R., *Surg. Gynecol. and Obst.*, 1932, lv, 265.
- ¹⁰ MCCANN, J. C., *Arch. of Surg.*, 1929, xix, 600.
- ¹¹ MORTON, C. B., *Ann. of Surg.*, 1928, lxxxvii, 401.
- ¹² SCHULLINGER, R. N., and STOUT, A. P., *Arch. of Surg.*, 1934, xxviii, 440.
- ¹³ WU, P. P. T., *Ibid.*, 1935, xxx, 557.

SHORT NOTES OF RARE OR OBSCURE CASES

LEIOMYOMA OF THE MEDIAN NERVE

BY CECIL P. G. WAKELEY

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TUMOURS of nerves are rare and always interesting to the surgeon inasmuch as they vary considerably in their symptomatology and often cause difficulty in diagnosis. The case described here was not diagnosed as a nerve tumour at all, and was thought to be a sarcoma of muscle. The following is the history of the case :—

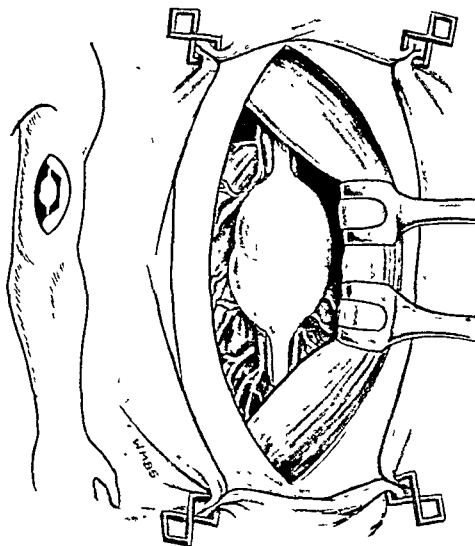


FIG. 168.—Drawing made at the time of the operation, showing the tumour situated on the median nerve. The inset shows the position of the incision in the arm.

Miss Emily D., aged 44, came under observation in October, 1935, complaining of a hard lump in the inner side of her left upper arm. She discovered the lump by accident about a year previously and it has slowly increased in size.

The swelling was hard but painless, and on palpation no painful sensations were produced in the forearm or hand. The size of the swelling was about that

of a tangerine orange ; it was fixed to the deep structures of the arm, but not to the skin, which could be freely moved over it. Although fixed to the deep structures it was not fixed to the humerus, and it did not limit any movements of the arm. Sensation in the whole upper extremity appeared to be normal.

Operation was decided upon, and on Nov. 21 a longitudinal incision was made over the tumour ; on incising the deep fascia it was found that the tumour was situated on the median nerve (*Fig. 168*). With some difficulty the tumour was completely excised from the nerve and all the small bleeding vessels were ligatured. The wound was closed with interrupted sutures.

An uninterrupted recovery ensued, with the exception of a slight paresis of the median nerve which has since completely cleared up. The excised tumour was quite smooth on the surface but had a very hard consistency ; on bisecting it a large area of central hæmorrhagic necrosis could be seen (*Fig. 169*). On histological examination the



FIG 169 —The tumour bisected, showing the area of necrosis in the centre

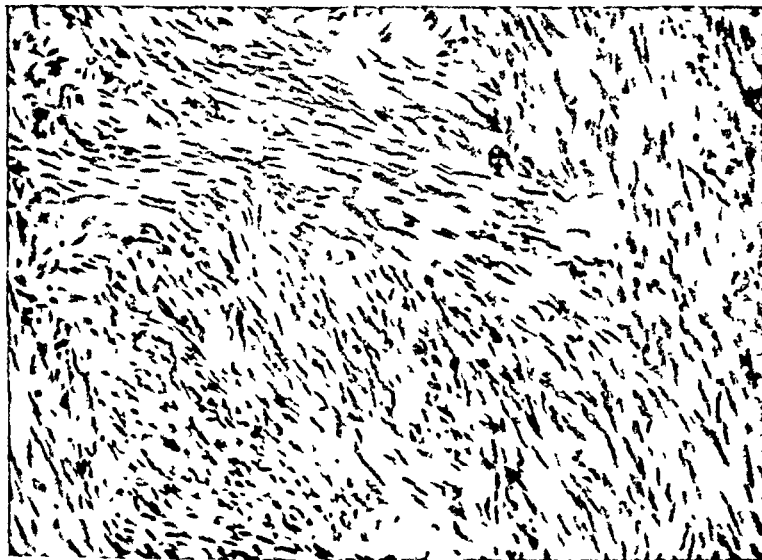


FIG 170 —Microphotograph showing typical arrangement of the leiomyoma.

tumour was found to be a leiomyoma. It consisted of bundles of parallel smooth muscle cells. The bundles showed some tendency to a whorled arrangement (*Fig. 170*). There were areas of œdema and of hyaline degeneration.

HÆMANGIOMA OF VOLUNTARY MUSCLE

By B. W. FRANKLIN BISHOP, KIMBERLEY

IN the October, 1935, issue of the JOURNAL there are two articles on the above condition. A further case is here recorded :—

HISTORY.—A Jewish youth, aged 19, came to see me complaining of a swelling in the lower part of his right leg. He had no idea how long it had been there, though his notice was drawn to it some two years previously. He could give no definite history of injury to that part of his leg. There was not much pain, though

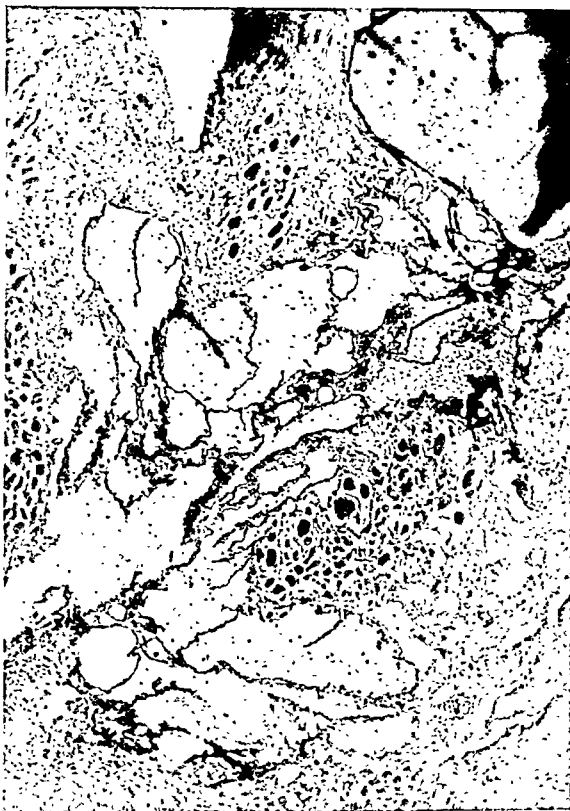


FIG. 171.—Microscopical section of hæmangioma of voluntary muscle.

he stated that his leg became tired easily and that he limped slightly. He could not say whether he had had this swelling all his life or not. He apparently did not attach much importance to it.

ON EXAMINATION.—There was an ovoid swelling about the size of a smallish orange situated in the lower third of his leg, posteriorly. It stopped short about

$\frac{1}{2}$ in. above his ankle. It seemed to be situated deeply in the muscles but was freely movable over the bone from side to side. It varied in consistency, parts appearing cystic and other parts hard. There was no pulsation either visible or on palpation. The posterior tibial artery was pulsating well. There was very definite wasting of the calf muscles, 1 in. difference as compared to the other leg. X rays revealed pressure indentation and erosion of the fibula, with minute areas of calcification in the soft tissues.

OPERATION.—A vascular dark-brown tumour was found implicating all muscles in the vicinity of the tumour. There was no capsule, and the tumour was so widespread that it was impossible to remove it *in toto*. An attempt was made to remove the margin. Portions were removed for section.

PATHOLOGICAL REPORT.—Spreading hæmangioma with islets of chronic inflammation and heterotopic bone.

Sections of the growth show the structure of a hæmangioma of the spreading type (*Fig. 171*). Foci of chronic inflammation and small islets of heterotopic bone are present in parts of the stroma.

Commentary.—In view of the rarity of these cases it seems of interest to report this example. It was impossible to state definitely from which muscle the tumour had arisen, though probably it was the peroneus longus.

RETROGRADE INTUSSUSCEPTION OCCURRING DURING LIFE

By ALEX. MITCHELL, ABERDEEN

IN view of a report on a case of retrograde intussusception occurring during life by Mr. Emlyn Lewis in the JOURNAL of January, 1936, I think the following case might be of interest.

A girl, aged 15, was seen on Feb. 12, 1935, with a history of intermittent abdominal pain for two days. During the last few hours the pain had become very severe. No definite physical signs were made out in the abdomen, except a little fixation and tenderness on the right of the umbilicus. Pulse and temperature were normal and the patient did not look ill. The bowels had acted two days previously after castor oil.

OPERATION.—Under a general anæsthetic the abdomen was opened by a right paracentral incision. Intussusception of the ileum about one foot from the ileo-cæcal junction was found. The intussusception was of a retrograde type and the enclosed portion was completely gangrenous. The portion of bowel affected was about one foot long. There was no appearance of tumour or any other abnormal condition to be made out. As the intussusception was irreducible, it was resected, with closure of both ends of the bowel and a lateral anastomosis.

The patient made an uneventful recovery and left the nursing home three weeks after operation.

The condition is undoubtedly a rare one, but this case alone is sufficient to show that it is not always a post-mortem condition.

ACUTE PSOAS ABSCESS CAUSED BY DIVERTICULITIS

BY ALEXANDER LYALL,

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THE complications of diverticulitis are many, and indeed it is one of the conditions which must be constantly kept in mind in the diagnosis of obscure intra-abdominal conditions. The following case illustrates yet another of its interesting complications, and one which, as far as we are aware, has not formerly been described.

John D., aged 64 years, a steelworks foreman, was admitted to the Glasgow Royal Infirmary on Jan. 17, 1935, with the following history. Seven weeks before admission the patient developed pain in the right leg. It was diagnosed at the time by his doctor as muscular rheumatism and he received massage treatment, which, however, made the condition much worse. The thigh became more swollen and very painful and he began to have sweating and feverish attacks. Upon admission to hospital, the right thigh was markedly swollen from the inguinal region down to the knee, where the swelling gradually faded away. The skin on the outer side of the thigh showed marked pitting on pressure. Under general anaesthesia incisions were made into the outer and the inner aspects of the upper thigh, and much foul-smelling pus was evacuated. Culture of the pus showed the presence of hæmolytic streptococci, and antiscarlatinal serum was given. The patient's condition gradually deteriorated, however, and he died six days after admission.

A post-mortem was performed ten hours after death. The body was that of a well-nourished elderly-looking man. Recent operation incisions were present on the outer and inner aspects of the right thigh, the external wound containing packing and exuding a great deal of pus. The sigmoid loop of bowel showed a well-marked diverticulosis and was adherent to the brim of the lesser pelvis on the right side. When these adhesions were broken down, a necrotic diverticulum was found to be attached to the psoas sheath and to communicate with an abscess in it. The pus extended upwards along the muscle to its lumbar attachment and downwards beneath the inguinal ligament into the thigh. Here the pus was found to have ruptured through the sheath and spread down the postero-internal part of the thigh to near the knee-joint. The pus surrounded the upper part of the femur and formed a large abscess in this region which had been opened by the operation incisions. There was no disease of the femur, ilium, vertebral column, hip, or sacro-iliac joints.

The condition was obviously an acute psoas abscess which had ruptured into the thigh and was caused by an inflamed diverticulum of the sigmoid becoming adherent to the sheath of the muscle.

My thanks are due to Mr. Macewen and Professor Shaw Dunn for permission to use the clinical and pathological summaries of this case.

REVIEWS AND NOTICES OF BOOKS

An Index of Differential Diagnosis of Main Symptoms. Edited by HERBERT FRENCH, C.V.O., C.B.E., M.A.(Oxon.), F.R.C.P.(Lond.), Consulting Physician to Guy's Hospital, etc. Super royal 8vo. Pp. 1145 + xii, with 742 illustrations (196 in colour). 1936. Bristol: John Wright & Sons Ltd. 63s. net.

It is eight years since the last edition of this work appeared, and the fact that since its first appearance in 1912 over fifty thousand copies have been distributed shows that it has been of great value. It really consists of a full descriptive text-book of medicine, surgery, and gynaecology, but all the information is arranged under symptoms and not under diseases. Its usefulness depends largely on the accuracy and completeness of the index, which occupies no fewer than 220 pages. Under the heading of pain in the back—for example—there are over ninety references in the index, and all other subjects are dealt with on the same scale. Over fifty pages are given to the diagnosis of 'swellings' in different parts of the body. The numerous illustrations are well chosen and well reproduced. Advances in recent methods of diagnosis of the biliary and urinary tracts are described, as well as the rarer diseases tularæmia, botulism, psittacosis, and pink disease. Dr. French, who has written the largest part of the book, is assisted by eighteen other contributors, of whom Mr. Ogilvie and Professor Bruce Perry take the place of the late Mr. Rowlands and Dr. Carey Coombs. It certainly forms an invaluable book of reference.

Handbook of Surgery. By ERIC C. MEKIE, M.B., Ch.B., F.R.C.S.E., Medical Officer, Malayan Medical Service. With a Foreword by JOHN FRASER, M.C., M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery, University of Edinburgh. Crown 8vo. Pp. 699 + xii, with 24 illustrations. 1936. Edinburgh: E. & S. Livingstone. 12s. 6d. net.

MR. MEKIE states the purpose of his book in the preface: "In such a handbook as this—expressly designed for the last-minute revision—little that is original can be incorporated except in arrangement, and all that is non-essential to the task of surmounting the final hurdle of the medical undergraduate career is excluded. The special subjects have therefore been omitted or rigidly abbreviated."

"In presenting this handbook I would urge the student to realize its limitations. It is but a supplement to his larger text-books, to which he must look for guidance on details and by which he should conduct his earlier studies."

The volume contains an enormous number of facts, which are clearly catalogued and in many instances are actually set out in tabular form. As one reads the book one is bewildered by the knowledge crammed into so small a space. For students revising for their qualifying examinations and also for the higher examinations in surgery, the book is excellent and should fill a most important place. One hopes—with the author—that it will be consulted only after the standard text-books have been read, and that it will not be used as the only means of acquiring a theoretical knowledge of surgery.

Elementary Surgical Handicraft. By J. RENFREW WHITE, Ch.M., F.R.C.S., F.A.C.S., Surgeon to the Dunedin Hospital, New Zealand, etc. Demy 8vo. Pp. 250 + vi, with 243 illustrations. 1936. London: J. & A. Churchill Ltd. 8s. 6d. net.

THIS small volume is produced in the hope that it may assist students and nurses in the handicraft of elementary procedures. The various methods of employing sutures, of tying knots, and of applying adhesive plaster strapping, are described with good illustrations. The pages devoted to the arrest of regional hæmorrhage will prove useful to house surgeons. The

book fulfils its mission so far as it clarifies many points which are often regarded as mysterious by the novice. In most respects we agree with the methods of approach to the uninitiated which the author adopts. The older methods of operating upon fractures and some detailed references to bone-grafting might with advantage be replaced by a description of local anæsthesia and the injection treatment of varicose veins and piles, and other minor procedures with which the student may be faced on receiving his final degrees. A revision of such a nature would be in keeping with the objects at which the author aims. It is perhaps unwise to emphasize in the preface of a book for students that surgery means handicraft. A good operator may be a menace to surgery unless he is also a first-class clinician, with instinctive judgement and alive to the necessity for strict personal attention to modern pre-operative and post-operative methods. The text, however, is confined to handicraft and contains many practical hints and suggestions.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc. With a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S.E. Third edition. Crown 8vo. Pp. 664 + xviii, with 648 illustrations. 1936. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE good reception given to this book has fully borne out what we expected after reading the first edition for review. It presents a much closer association of anatomy with practical surgery than any other book we know. The section, for example, on the sympathetic, with the diagrams explaining the various operations, is a model of clearness. The present edition has a detailed description of the nerves related to the thyroid gland, of renal-adrenal adherence, and diaphragmatic hernias. There have also been numerous additions to the diagrams which make so much for the elucidation of the text.

Traité de Chirurgie d'Urgence (Félix Lejars). By PIERRE BROCCQ, Professor agrégé à la Faculté de Médecine de Paris; Chirurgien des Hôpitaux; and ROBERT CHABRUT, Ancien Chef de Clinique à la Faculté de Médecine de Paris. Ninth edition. Large 8vo. One volume of 1286 pages, with 1250 illustrations. Paper covers, Fr. 170; bound, Fr. 190. In two volumes (Pp. 1299 + xv), Fr. 200. 1936. Paris: Masson et Cie.

LEJARS' *Chirurgie D'Urgence* first appeared in 1899; eight editions have already been published in France; it has been translated into many languages, and many thousands of copies have been sold throughout the world. A book with such a record has become an institution, no longer subject to the ordinary critical review; a new edition was anticipated with pleasure and welcomed on its appearance.

Chirurgie D'Urgence was Félix Lejars' master work. It was the expression of his surgical life. He said that he had lived it before he wrote it, had produced it slowly and painfully, dominated by a burning desire to lighten the burden of suffering for the sick and the injured and at the same time to help the practitioner of medicine, particularly the isolated practitioner, the hospital resident, and the young surgeon. It was with these ideals and in this spirit that the eight editions were written. Amongst the last desires of a strenuous life, Lejars had contemplated the preparation of a ninth edition. Weariness primarily, sickness subsequently, prevented the realization. It was necessary to entrust the work to other hands. The new edition appears with the names of Professor Pierre Brocq and Dr. Robert Chabrut on the title page.

When one has known the book since its first appearance and known its author, and has followed it carefully and appreciatively through the later editions and impressions, some feeling of anxiety is unavoidable when greeting the new volumes, lest it should be found that past high standards and traditions have not been adequately maintained. Let it be said at once, close and critical examination shows that there is no ground for any fears.

The present authors claim that though the book has been entirely recast and brought into touch with the very latest surgical thought and practice, they have closely followed the original plan and have striven earnestly to maintain the ideals and spirit which characterized the previous editions.

Notable changes have come about in recent years in the conditions under which medical and surgical work is carried on. Particularly is this the case with regard to means of transport

and the provision of new, adequately equipped, and well-staffed hospital centres, even in remote and sparsely populated districts. It now but rarely happens that a practitioner is left, dependent on his own resources, isolated except perhaps for the assistance of an anaesthetist, face to face with a serious surgical emergency. But though he is absolved from the necessity of actually performing the operation or carrying out the necessary treatment, though it may be easily possible to obtain skilled advice and help, though it may be a simple matter to dispatch the patient to the nearest surgical centre, it is even more important than before that he should be able to recognize early signs and symptoms and indications and be fully alive to the possibilities and limitations of surgery. Satisfactory results in urgent conditions depend largely on early intervention, which again depends on early diagnosis and action on the part of the practitioner who first sees the patient.

Following the lines laid down originally by Lejars, urgent surgical conditions are envisaged in the very widest possible sense. The book is no mere manual of minor surgery; though the needs of the general practitioner and the hospital resident are fully considered and amply provided for, it is perhaps to the young, well-trained, but relatively inexperienced general surgeon that it will make the greatest appeal and be of the greatest use. Incidentally it may be added that even the experienced surgeon will find much of value and interest. The chapters dealing with the treatment of fractures of the base of the skull, with intrathoracic wounds, injuries, and suppurations, with thoraco-abdominal lesions, and with subphrenic and hepatic abscesses are well worthy of careful consideration, together with much else.

The subject matter has been so very well brought up to date that here and there are to be found suggestions and recommendations so novel that they can hardly be accepted forthwith. Attention may particularly be directed to the chapter on the Prevention of Tetanus, which is based upon and embodies textually the Report of a Commission appointed by the Society of Surgeons of Paris to study the question. The report, dated July, 1935, received, and has since been published with, the approval of the Society. Shortly, it condemns the use of antitetanic serum except for treatment, and strongly advocates active immunization by means of antitetanic vaccination, particularly of conscripts called up for military service and of workers in certain occupations carrying risks of repeated minor injuries.

It is needless to particularize further. The same high standard is maintained throughout; the ardent spirit of Félix Lejars still pervades the pages. One may not always agree, but one must always think. *Chirurgie D'Urgence* has been and remains a great book. It is unique. To any medical man or woman, practitioner or specialist, physician or surgeon, who can read French it is to be recommended; even those who have no great knowledge of the language may follow the text in the wealth of illustrations, well-planned, well-executed, and informative. An excellent index of illustrations is a valuable, and to the reviewer a novel, feature of the work.

Post-graduate Surgery. Edited by RODNEY MAINGOT, F.R.C.S., Senior Surgeon to the Royal Waterloo Hospital, etc. In three volumes. Royal 8vo. Vol. II. Pp. 1747-3572, with 1134 illustrations. 1936. London: Medical Publications Ltd. £9 9s. the set, or £3 10s. per volume.

THE expectations aroused by the first volume of this great work have been more than fulfilled by the second. First, as to the actual contents of this volume, the mere enumeration of which is a matter of some length.

The section on the Head, Spinal Column, and Salivary Glands by Cecil Wakeley is a remarkable combination of simple, practical instruction with scientific exposition of the subject. (But why speak of 'Spinal Column' when the subject is that of the Spinal Cord?) The routine examination in cases of head injury is very clearly put. The illustrations of operations on the Gasserian ganglion and its root are beautifully clear. The whole question of tumours of the brain is made simple by the very concise descriptions and figures.

The chapters on the Neck are by Sir William Wheeler, and include a very well illustrated account of the excision of thyroglossal sinuses, and also of Henry's operation for the ligation of the first part of the subclavian artery.

McNeill Love writes on the Surgery of the Breast. He advocates and illustrates a good method of 'closed' drainage of a mammary abscess. In the description of the radical breast amputation we think that the advantages of the use of the diathermy knife might have been mentioned.

The chapters on the Thorax are an example of one of the best features of this work, because they embrace the medical as well as the surgical aspects of the subject. They are written by Sleigh Johnson and Holmes Sellors, and include post-operative chest complications, artificial pneumothorax, empyema, the surgical treatment of pulmonary tuberculosis, pulmonary embolism, and bronchiectasis. We should have thought that a work of this size might have included some account of the surgery of the heart, but perhaps this is to come in the third volume.

The section on Gynæcology is by Lyle Cameron (regional gynæcology and gynæcological operations), Sidney Forsdike (sterility), and V. B. Green-Armytage (ovary). It represents a very complete treatise on gynæcology, including an account of the investigation of the causes of sterility by radiology and other methods, and the various operations done for the relief of certain types of this condition.

Ainsworth-Davis writes on the Urinary System and the diseases of the male genital organs. This section begins with eighty-three pages devoted to the various physical and biochemical methods of examination, including the routine examinations—cystoscopy, pyelography, etc.—and the less common methods of urethroscopy and vesiculography. In relation to the diseases of the kidneys, a long account is given of the ketogenic diet, but we are told that the use of mandelic acid has now superseded this. A full account is given of von Lichtenberg's operation for the plastic repair of hydronephrosis. In the section on the prostate full accounts are given of the Harris operation for repair of the prostatic bed and of the perurethral operation. In the treatment of retained testis Ombrédanne's method of orchidopexy, in which the testis is merely anchored in the scrotum, is the only one given, and we think that the more efficient method of anchoring the testis and scrotum to the thigh (Torek) should have been mentioned. In the operation for excision of urethral stricture, no mention is made of the longitudinal splitting of the tube, a point the importance of which was demonstrated by Hamilton Russell.

A special part is devoted to the Sympathetic Nervous System, its anatomy, physiology, and clinical aspects. This is by Lawrence Abel, and is most clear and thorough, with a wealth of clear illustrations. The chapter on methods of investigation in relation to the diagnosis, best mode of treatment, and prognosis, is of great value, as is that on the different operations, with a critical account of their value.

Adrenal gland surgery (Broster) and pathology (Vines) represent an interesting phase of new surgical work. To this section is relegated the only coloured plate in the book, which shows the special reaction of normal and abnormal tissue to the fuchsin stain.

A whole section of the book is devoted to what is termed 'Injection Therapy'. This includes the treatment of hæmorrhoids and varicose veins (Harvey), hydrocele and varicocele (Maingot), and chronic gravitational ulcers of the leg. We are surprised that it should be deemed wise or necessary to describe the injectional treatment of varicocele and hydrocele. But our surprise is still greater to find the injectional treatment of hernia seriously advocated by Delisle Gray.

A short chapter on Infections of the Hand is by Hamilton Bailey. The teaching fully adopts Kanavel's work, and is well illustrated by many clear diagrams.

The last section is on 'Orthopædics' and is by St. John Buxton. We think that some explanatory note should introduce this article to explain the curious way in which the subject matter has been chosen. 'Deformities of the feet' include no account of ordinary club-foot (equino-varus); 'disabilities of the knee-joint' make no mention of injuries of the crucial ligaments or of genu valgum or varum; 'acute arthritis' is discussed, but not chronic. Open fractures are 'orthopædic', but not the simple ones; neither deformities of the spine, congenital dislocation of the hip, nor poliomyelitis find any place in this section, and we are left to suppose that they will be dealt with elsewhere.

As we said in reviewing the first volume, the work is of outstanding merit, and a great tribute to the energy and scientific acumen of its many authors. The printing is large and clear, and the illustrations in black and white lavish in the extreme. In fact we think that the number of figures is excessive. It is unnecessary to have ten illustrations of parotid tumours, and many figures from instrument makers' catalogues, e.g., a 'cystoscopic' stool and a stand for a diathermy apparatus, are superfluous. We sincerely trust that the contents of the last volume will allay our apprehensions about the matters of bone and joint surgery.

Minor Surgery and the Treatment of Fractures (Heath: Pollard: Davies). By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Twenty-first edition. Crown 8vo. Pp. 485 + viii, with 284 illustrations. 1936. London: J. & A. Churchill Ltd. 10s. 6d. net.

A book which first appeared in 1861, and which has now reached its twenty-first edition, hardly needs any commendation.

We are glad to note, however, that the present edition has acknowledged its first founder, Christopher Heath, by giving a frontispiece portrait and an epitome of his founding this work. The art of bandaging no longer finds a place in the title, but is adequately described and illustrated. Fractures of the long bones and of the spine are well described, and due prominence given to the modern methods of splinting by plaster-of-Paris. We think that an inclusion of Cramer's wire splinting would have been useful. The clearness of the line drawings leaves nothing to be desired.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London, etc. Fifth edition. Large 8vo. Pp. 287 + xii, with 341 illustrations. 1935. Bristol: John Wright & Sons Ltd. 21s. net.

In his Ramon Guiteras Lecture delivered in 1932, Henry Wade said, "The wards are the greatest of all research laboratories." This statement is used as a motto for this new edition, and the whole book is a tribute to the value of the clinical work which it enjoins. Nearly all the illustrations are original and most of them are excellent, but it would be an advantage to have still more of the telling diagrams copied from the author's case notes. It seems strange that the book still lacks an adequate illustration of Dupuytren's contracture and of the malignant umbilical nodule.

It is almost unnecessary to say anything in recommendation of this well-known work, though it is a pleasure to record once again that it fully maintains the very high standard of its predecessors.

Urology in Women: A Handbook of Urinary Diseases in the Female Sex. By E. CATHERINE LEWIS, M.S.(Lond.), F.R.C.S., Surgeon to the Royal Free Hospital; Surgeon and Urologist to the South London Hospital for Women. Second edition. Demy octavo. Pp. 100 + viii, with 31 illustrations. 1936. London: Baillière, Tindall & Cox. 6s. net.

THIS book was reviewed in this Journal in 1932, and we congratulate the author on the well-deserved compliment implied by the need for a second edition.

In this new edition sections have been added on obstruction of the vesical neck in women and on endometriosis. In addition, some of the accounts of treatment have been amplified in the light of further experience, and lastly there is a full description of the changes that occur in the ureters during menstruation and pregnancy, which is based largely on the work of Baird.

One of the most interesting sections is that on radium injuries to the bladder; these have been known to ensue on the use of radium for non-malignant affections of the uterus, such as fibroids. The attention of both gynaecologists and radium experts should be directed to preventing these ulcers, which may be the source of danger as well as of great inconvenience to the patient. The author points out that their presence may not be detected until many months after the original treatment; in one case the patient nearly lost her life through hæmorrhage two years after the use of radium for menorrhagia.

No mention is made of the use of mandelic acid in the treatment of chronic bacillus infection, though the author lays emphasis on the difficulty some patients experience in persevering with the ketogenic diet; we should have liked to have read her opinion on this.

One statement is open to criticism; on page 95 she states that hydronephrosis is the commonest complication of nephropotosis; in view of recent research on this condition, we should like to know whether this unqualified statement springs from her own experience.

With the exception of one misprint—on page 42 'tongue' should obviously be 'trigone'—the book is admirably turned out; the illustrations are good, and there is a useful and not too voluminous bibliography.

Diagnostic Roentgenology. Edited by ROSS GOLDEN, M.D., Professor of Radiology, the College of Physicians and Surgeons, Columbia University; Director of the Department of Radiology, Presbyterian Hospital, New York. $9\frac{1}{2} \times 7\frac{1}{2}$ in. Pp. 867 + xi, with 964 illustrations. 1936. New York and Edinburgh: Thomas Nelson & Sons. \$20.00.

THIS is a very complete work on diagnosis by means of X rays. The following quotation from the Editor's Preface gives a good idea of the underlying principle which pervades the work: "The information obtained from an X-ray examination, even when it points clearly to a definite pathologic diagnosis, must be correlated with the history, the physical examination, laboratory findings, etc. This can best be accomplished by the co-operation of clinician and radiologist. The former must not expect his clinical impression always to be confirmed, and must be prepared at times to discard certain preconceived ideas in regard to a case; the latter, while considering the clinical evidence and 'what ought to be there', must at the same time keep an objective and unbiased point of view to avoid overlooking something unsuspected by the clinician. The importance of cordial sympathetic co-operation of clinician and roentgenologist and frank discussion of specific clinical problems cannot be over emphasized."

The book is thus much more than a mere X-ray atlas, although it is indeed a wonderful collection of roentgenographic records. Every section consists of a discussion of the problems of diagnosis and pathology of each organ or system, illustrated by typical radiograms. The skull; the accessory sinuses; the chest wall; the lungs, pleura, bronchi; the heart and great vessels; the stomach, intestines, liver, gall-bladder; the bones, normal and pathological, at all ages; the joints; the spine, spinal cord tumours; the kidneys, ureters, and bladder; the uterus and Fallopian tubes; the bony pelvis and certain aspects of fractures which may cause difficulty in diagnosis: all are treated fully and clearly.

Obviously this book will form an invaluable work of reference for physicians, surgeons, and gynaecologists.

Tumours of the Urinary Bladder. By EDWIN BEER, M.D., F.A.C.S., Visiting Surgeon, Mt. Sinai Hospital; Consulting Surgeon, Bellevue Hospital, New York City. Large 8vo. Pp. 166 + vi, with 52 illustrations (8 in colour). 1925. London: Baillière, Tindall & Cox. 16s. net.

THIS monograph deals with the pathology, the diagnosis, and, more particularly, the treatment of bladder tumours; as such, it is a valuable contribution towards the elucidation of a very obscure and difficult problem of surgery.

The author speaks with authority, for he and his colleagues at the Mount Sinai Hospital have dealt with some hundreds of these tumours, and they have the advantage of a very efficient 'follow-up department'.

Undoubtedly the most important conclusion reached by Dr. Beer is that, in spite of an extensive trial of radium and deep X-ray therapy, treatment of malignant vesical tumours by surgical excision is the most satisfactory.

There are several points of interest in the chapter on the destruction of papillomata by diathermy: no anæsthetic is used in such cases, as pain implies a burn of the bladder wall and thus acts as a danger signal to prevent a traumatic perforation of the viscus; an unsatisfactory result after diathermy should arouse a strong suspicion that the papilloma is malignant; lastly, tumours at the bladder neck are treated by the resectoscope and not by the use of a retrograde cystoscope—indeed, we are surprised to find no mention of Swift Joly's beautiful instrument.

The technique employed in partial resection of the bladder is similar to that used by English surgeons; there is emphasis laid on one point of the operation, and that is that the tumour should be examined from the outer side of the bladder in order to determine the amount of infiltration before deciding on the scope of the excision. The author has found, contrary to the experience of research workers, that there is no marked increase in the bladder capacity following a partial resection; and that oblique implantation of the ureter into the bladder wall is difficult and quite unnecessary, a finding which is confirmed by the observation of kidney function some time after implantation, for intravenous pyelography shows little impairment.

There is an excellent account of total cystectomy, of which no less than eighteen cases are described; of these, five died and six are apparently cured. Dr. Beer advocates bringing the ureters out through the skin close to the anterior superior spines; he is the first to acknowledge that it is an unpleasant business for the patient, but he points out that the mortality of a preliminary implantation into the bowel varies from thirty to fifty per cent.

The illustrations are good, and there is a bibliography; we congratulate the author on a first-class piece of work, and cordially commend the book to our readers.

Die Chirurgie der Hirngeschwülste. By Prof. Dr. N. GULEKE, Direktor der chirurgischen Univ.-Klinik, Jena. Part 5 of *Vorträge aus der praktischen Chirurgie*, edited by Prof. Dr. ERICH LEXER (Munich). Royal 8vo. Pp. 40. 1936. Stuttgart: Ferdinand Enke. RM. 2.20.

THIS small monograph gives Professor Guleke's experience of tumours of the brain and their treatment, over a considerable period. He considers that ventriculography and arteriography allow a local diagnosis in a very high percentage (94 per cent) of tumours. Meningiomas, tumours of the hypophysis, acoustic nerve tumours, and benign gliomas can be removed entirely. For malignant gliomas in elderly subjects, and in medulloblastomas in children, a decompression operation followed by X-ray therapy should be adopted. There seems to be no success with X-ray therapy without operation. A cerebral tumour should be operated upon as soon as the diagnosis is established, in order to avoid such complications as sudden increase of pressure, hæmorrhage into the ventricles, and blindness. Radical operation of the tumour should be done if possible. Partial removal has the advantage of giving more room to the brain and tumour, but malignant change may occur later.

Local anæsthesia should be employed in all operations for tumours of the cerebrum. To avoid fluctuation of pressure during operation, hypertonic solutions of glucose are given intravenously, and magnesium sulphate rectally, as well as puncture of the ventricles. The dura is opened very slowly and the tumour is removed by the electric loop. These operations can be done in one stage, except in cases where there are dangerous fluctuations of blood-pressure. It is possible, nowadays, to remove not only tumours of the ventricles and their neighbourhood, but also cysts situated between the ventricles. Tumours of the hypophysis should be removed transfrontally.

Professor Guleke considers that the prognosis of operations on the brain has greatly improved during the last twenty years. The mortality has decreased from 40-80 per cent to 20-30 per cent, whilst Cushing has a mortality of only 16.6 per cent. In one-third of operated cases a lasting cure has resulted.

The Study of Anatomy. Written for the Medical Student. By S. E. WHITNALL, M.A., M.D., B.Ch.(Oxon.), M.R.C.S., L.R.C.P., F.R.S.(Canada), Professor of Anatomy in the University of Bristol. Third edition. Crown 8vo. Pp. 113. 1936. London: Edward Arnold & Co. 4s. 6d. net.

THIS little book, which costs only four shillings and sixpence, is surely well worth digestion by any medical student. The title is the worst part of it—*The Study of Anatomy*. This makes one think that it is a pocket text-book. It is not the least like that, but rather a brief philosophic talk of a friendly and human kind. Osler would have loved it. It breathes his spirit. Need we say more?

Les petites Règles de la Chirurgie parfaite. By J. OKINCZYC, Professeur agrégé; Chirurgien des Hôpitaux de Paris. Royal 8vo. Pp. 60. 1936. Paris: Masson et Cie. Fr. 12.

THIS pamphlet in French is in the form of a lecture sermon to a new house surgeon. Rather didactic in character, it tells him how to assist at an operation, how to dab, how to tie knots, how to use a Reverdin needle, how to retract, and how to stitch up a wound. Simple notes are laid down for pre- and post-operative care. It is full of useful tips not always found in a text-book, and the style, though colloquial, is occasionally witty.

Gefäßmissbildungen und Gefäßgeschwülste des Gehirns. By Prof. Dr. HILDING BERGSTRAND (Stockholm); Prof. Dr. HERBERT OLIVECRONA (Stockholm); and Prof. Dr. WILHELM TÖNNIS (Würzburg). Large 8vo. Pp. 181, with 137 illustrations. 1936. Leipzig: Georg Thieme. Paper covers, RM. 24; bound, RM. 26.

VASCULAR tumours of the brain are being discovered more frequently each year in the various neurological clinics both at home and abroad. The present monograph is the outcome of the investigations of clinical and pathological material by Professors Bergstrand, Olivecrona, and Tönnis, in Stockholm. The clinical and pathological material is well set forth, and the illustrations are excellent, there being some 137 of these. The arteriograms are very instructive and clearly show the value of injection therapy in the diagnosis of vascular tumours of the brain. There is an excellent bibliography, which contains nearly 400 references, and is to be found at the end of the monograph. Vascular tumours of the brain represent some 2 per cent of all brain tumours, and in the past must have been missed in most of the clinics.

This monograph forms a useful companion to that published by Harvey Cushing in 1928.

Lehrbuch der Kriegschirurgie. By Dr. CARL FRANZ, Honorarprofessor der Universität, Berlin; etc. Second edition. Large 8vo. Pp. 424 + vii, with 121 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 26.60; bound, RM. 28.80.

IT is a matter of some significance that this text-book of military surgery, which was first published in 1919, should now appear in a second edition. The preface to this edition points out that conferences, leagues, and pacts have failed to prevent wars, and that therefore military surgery must be kept up to date.

An excellent feature of the book is the recognition given to methods of treatment used by all the nations engaged in the Great War. The value of the various patterns of Thomas splint both for transport and treatment of fractures is fully recognized and illustrated. It is remarkable to note what a large proportion of space is devoted to the injuries of the limbs, the blood-vessels, and the nerves, whilst the gunshot wounds of the abdomen are compressed into sixteen pages. One is tempted to wonder whether there is any English book ready for publication which would summarize and standardize military surgery in readiness for the next war.

The Human Foot: its Evolution, Physiology and Functional Disorders. By DUDLEY J. MORTON, Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Large 8vo. Pp. 244 + xvi, with 100 illustrations. 1935. New York: Columbia University Press. London: Oxford University Press (Humphrey Milford). 15s. net.

THIS book has been written from an aspect that will be unfamiliar to many who concern themselves with the treatment of foot disorders.

The first part of the book, devoted to the evolutionary development of the foot, consists of a critical survey of the problem of the terrestrial locomotion of living organisms, and the stages which it is assumed have led to the development of the arched human foot as we know it. Though chiefly concerned with the skeletal changes, the author deals also briefly with the muscles.

The second part deals with the physiology of the foot both of civilized and primitive communities. The author considers the foot with regard to its function of balance and weight supporting, and later as an agent of locomotion. It will be found that he gives greater prominence to the part played by the ligaments in arch support than will be accepted by all authorities, and produces interesting evidence in criticism of the widely accepted conception of an anterior transverse arch.

The third part of the book describes certain functional disorders which have come to the author's notice. As the preceding chapters show that the arched foot is a human characteristic which has been acquired slowly and laboriously throughout the development of the race, it is not surprising that these functional defects concern imperfections of this arch structure.

His analysis is based on data derived from clinical investigation, X-ray evidence, and records made by his staticometer and kinetograph. The main defects he finds are: short first metatarsal bone, posteriorly placed sesamoid, and hypermobility of the first metatarsal segment. He concludes by considering the principles of treatment and the methods which can be used to produce correction of weight distribution.

The book is based on the results of careful original investigation and will be of great value to anatomists and orthopædic surgeons.

Hero-Dust. By JAMES KEMBLE, Ch.M., F.R.C.S. Pp. 193 + xvi, 1 illustration. 1936. London: Methuen & Co. Ltd. 6s. net.

MR. JAMES KEMBLE, the author of *Idols and Invalids*, now contributes an equally readable volume which he calls *Hero-Dust*. It is written primarily for laymen, but contains much that will give thought to medical men, for Mr. Kemble has read widely and is able to throw light upon his problems by his knowledge of medicine and surgery. The longest and perhaps the most important essay deals with the blindness of John Milton; from the details given it is clear that it was due to chronic glaucoma attacking first his left, and some three years later his right, eye. Dealing with Catherine the Great, the author tells the story of her inoculation with small-pox by Dr. Dimsdale, one of whose descendants became Lord Mayor of London. He tries also to unravel the paternity of her children, but without complete success, as she is known to have had twelve lovers from the time when she was 23 until she reached the age of 60. The troubles of Mary Queen of Scots he considers from a new angle; he believes that she was with child by Bothwell before she married him, and that she was six months' pregnant when she miscarried. Beau Brummell and Omar Khayyâm conclude a little volume which will pass away a leisure hour with pleasure and profit.

My Life and Work: The Search for a Missing Glove. By Dr. ADOLF LORENZ, Hofrath and Professor of Orthopædic Surgery, University of Vienna. Demy 8vo. Pp. 362 + xi. Illustrated. 1936. New York and London: Charles Scribner's Sons Ltd. 12s. 6d. net.

DR. ADOLF LORENZ tells in this autobiography how from nothing he rose to be Professor of Orthopædic Surgery in the University of Vienna and was complimented with the title of Hofrath. He tells the story modestly and well, speaking of his early struggles, of the reasons which led him to adopt orthopædic surgery, and of his success, more especially in the treatment of congenital displacement of the hip. Many will still remember the demonstrations he gave at Liverpool when on his way to visit the United States for the first time, and of the number of imitators he had in this country. But he says comparatively little of the surgical side of his work. The book is chiefly personal, and as such is extremely good reading, for it is the life history of one who became, as he always intended to be from his infancy, 'ein grosser Herr'. From the depths of poverty he attained to wealth, and from riches he sank again into poverty, as Austria was ruined by the war and he had invested his savings in home securities. Nothing daunted, he set to work again in the United States, and by his appeals was able to send help to the many children whom he knew were starving in Vienna. Prof. Lorenz paints himself as an optimist, somewhat of a mystic with an inherent dread of snakes, a gourmet always longing for his native dishes, with a peasant's hunger for land and yet with a love for building and pretty surroundings. He has been fortunate in securing a wife who acted for many years as his assistant in practice, who lives happily with him, and curbs his tendency to extravagance. There is an excellent index and the illustrations are well reproduced.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

The Operations of Surgery. By R. P. ROWLANDS, M.S. (Lond.), F.R.C.S., late Surgeon to Guy's Hospital, etc.; and PHILIP TURNER, B.Sc., M.S. (Lond.), F.R.C.S., Consulting Surgeon to Guy's Hospital, etc. Eighth edition. Volume I: The Upper Extremity. The Head and Neck. The Thorax. The Lower Extremity. The Vertebral Column. Royal 8vo. Pp. 1495 + x, with 435 illustrations (38 in colour). 1936. London: J. & A. Churchill Ltd. 36s. net.

Manual of Emergencies: Medical, Surgical and Obstetric: Their Pathology, Diagnosis and Treatment. By J. SNOWMAN, M.D., M.R.C.P.Lond. Third edition. Crown 8vo. Pp. 401 + ix. 1936. London: John Bale, Sons & Danielsson Ltd. 10s. 6d. net.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. Second edition. Fcap 8vo. In two volumes. Pp. 180 + viii, with 37 illustrations. 1936. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each net.

Die Nachbehandlung nach Operationen: ein Lehrbuch in Vorlesungen. By Prof. Dr. PAUL REICHEL, Geheimer Sanitätsrat. Third edition. Large 8vo. Pp. 499 + xii, with 85 illustrations. 1936. Munich: J. F. Bergmann. Paper covers, RM. 24; bound, RM. 25.80.

Roentgenographic Technique: A Manual for Physicians, Students, and Technicians. By DARMON ARTELLE RHINEHART, A.M., M.D., F.A.C.R., Professor of Roentgenology and Applied Anatomy, School of Medicine, University of Arkansas, etc. Second edition. Large 8vo. Pp. 431, with 183 illustrations. 1936. London: Henry Kimpton. 28s. net.

Cystoscopy and Urography. By JAS. B. MACALPINE, F.R.C.S., Hon. Surgeon and Surgeon in Charge of the Genito-urinary Department, Salford Royal Hospital, Manchester. Second edition. Large 8vo. Pp. 478 + xv, with 297 illustrations and 14 coloured plates. Bristol: John Wright & Sons Ltd. 30s. net.

Orthopædic Surgery. By WALTER MERCER, M.B., Ch.B., F.R.C.S.(Edin.), F.R.S.(Edin.), Assistant Surgeon, Royal Infirmary, Edinburgh, etc. With a Foreword by JOHN FRASER, M.C., M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh. Second edition. Medium 8vo. Pp. 906 + xi, with 408 illustrations. 1936. London: Edward Arnold & Co. 40s. net.

A Handbook of Urology for Students and Practitioners. By VERNON PENNELL, M.A., M.B., B.Chir. (Cantab.), F.R.C.S. (Eng.), Hon. Surgeon and Surgeon with Charge of Urological Department, Addenbrooke's Hospital, Cambridge; etc. Cr. 8vo. Pp. 224 + viii, with 34 illustrations. 1936. London: Cambridge University Press. 7s. 6d.

Pathology of the Nervous System. A Student's Introduction. By J. HENRY BIGGART, M.D. (Belfast), Pathologist to the Scottish Asylums' Board; Neuropathologist to the Royal Infirmary, Edinburgh; etc. With a Foreword by Professor A. Murray Drennan, M.D., F.R.C.P. Demy 8vo. Pp. 335 + xvi, with 204 illustrations. 1936. Edinburgh: E. & S. Livingstone. 15s.

A Descriptive Atlas of Radiographs. An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Third Edition, revised and enlarged. Cr. 4to. Pp. 560 + xxxi, with Frontispiece and 794 illustrations. 1936. London: Henry Kimpton. 42s.

- La Pratique Chirurgicale Illustrée. Fascicule XX.** Large 8vo. Pp. 259, with 219 illustrations. 1936. Paris: G. Doin et Cie. Fr. 70.
- Le Cancer (Etudes Anatomico-Cliniques).**—By I. STOIA, Docent de la Faculté du Médecine de Bucarest; Médecin chef du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc.; and P. STANCIULESCU, Assistant du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc. With a Preface by Professor CH. LENORMANT. Super royal 8vo. Pp. 322, with 147 illustrations. Paris: Masson et Cie. Fr. 55.
- Fasciæ of the Human Body and Their Relations to the Organs They Envelop.** By EDWARD SINGER, M.D., Department of Anatomy, College of Physicians and Surgeons, Columbia University. 10½ / 7¼. Pp. ix + 105, with 24 illustrations. 1935. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 13s. 6d. net.
- Studies on the Healing of Fractures with Special Reference to the Significance of the Vitamin Content of the Diet.** By JOHN HERTZ, M.D., Assistant Surgeon, Department C, Surgical University Hospital, Copenhagen. Super royal 8vo. Pp. 286, with 82 illustrations. 1936. Copenhagen: Levin & Munksgaard. (London: Oxford University Press.) Kr. 15.
- Roentgendiagnostik der Knochen- und Gelenkkrankheiten.** By Professor Dr. ROBERT KIENBÜCK (Vienna). Part 4. Degenerative Wirbelsaulenerkrankungen. Large 8vo. Pp. 229–450, with 214 illustrations. 1936. Berlin and Vienna: Urban & Schwarzenberg. RM. 24.
- A Short Practice of Surgery.** By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London, etc.; and R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon, Royal Northern and Metropolitan Hospitals, etc. Third edition. Demy 8vo. Pp. 995 + viii, with 996 illustrations, of which 88 are coloured. 1936. London: H. K. Lewis & Co. Ltd. 28s. net.

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BY SIR D'ARCY POWER, K.B.E., LONDON

XI. TWO LIVERPOOL SURGEONS

I. HENRY PARK, WHO EXCISED JOINTS IN 1781

HENRY PARK, surgeon to the Liverpool Hospital, was amongst the first to recommend excision of joints when amputation was the routine method of treatment, and his work is therefore well worthy of remembrance. For the facts of his life as well as for his portrait I am indebted to Mr. Raymond Tinné Berthon, his great-grandson.

Henry Park was born in Water Street, Liverpool, on March 2, 1744-5, the son of Edward Park, apothecary, and Mary, daughter of John Lyon, mariner, of the same town. He was apprenticed first to James Blomfield, surgeon to the Liverpool Infirmary, his uncle by marriage; afterwards to Percivale Pott, with whose family he maintained so close a connection that Edward Holden, Pott's second son, is said to have been buried in Park's family grave at St. James's, Toxteth Park, Liverpool. This Edward Holden Pott is described as "late Major of the Royal Westminster Militia who died September 1st, 1798, aged 32 years."

Pott's influence on Park is shown by the pamphlet published in 1783 with the title, "An account of a new method of treating Diseases of the Joints of the knee and elbow in a letter to Mr. Percival Pott." In this letter he describes a case of excision of the knee for advanced tuberculous disease in the following words. "I had under my care in the Infirmary Hector M'Caghen, a strong, robust Scotch sailor, aged thirty-three, who was admitted for a diseased knee of ten years standing, the joint, though pretty considerably enlarged, was by no means so much so as is frequently met with in scrophulous affections; yet the integuments were so tense as to appear incapable of yielding to further distension; the contraction of the Flexor muscles was such as to draw back the leg, so as to form a right angle with the thigh, in which position it was immoveably fixed; apparently some degree of union with the bones had begun to take place, but this could not yet be determined with certainty, as every attempt to communicate to the joint the smallest degree of motion gave him the most excruciating pain. This poor man's sufferings were daily increasing and his health daily declining, in such a degree, that he began to beg to have the limb taken off. This, however, I could not consent to do, without first proposing

and explaining to him the extirpation of the joint, in order that he might have the chance of such a cure, if he chose to take it. He assented without much hesitation and the operation was accordingly done on the 2nd July, 1781."

Park had previously performed excision of the knee and elbow on dead bodies, using a longitudinal incision and removing the patella. He found, however, that he "had not made sufficient allowance for the difference between a healthy and a diseased state of the parts ; in short, there appeared so much confusion of parts on opening the articulation, the ligaments being in some parts extremely thickened and



HENRY PARK

horny, in others in a sloughy suppurated state with the cartilages almost wholly destroyed and the heads of the bone much eroded by the offensive matter of which there was a good deal in the joint ; besides that some degree of bony union had begun to take place between the head of the tibia and the inner condyle of the Femur that, after spending some time in the attempt and consequently making the operation much more painful and tedious it was thought advisable to relinquish this idea and to make a transverse incision and divide the femur above the condyles.

"The quantity of bone removed was somewhat, though not much, more than two inches of the femur, and of the tibia rather more than one inch which were but

just enough to enable me to bring the leg into a right line with the thigh. The only artery that was divided in the operation was one on the anterior part of the knee which ceased to bleed before the operation was concluded though the pulsation continued pretty strong in the ankle; the ends of the bone, however, particularly that of the femur bled pretty freely. A few stitches were passed through the skin, as well along the course of the Transverse incision as of that part of the longitudinal one that extended up the thigh. The lightest superficial dressings only were applied and the limb placed in a case of tin sufficiently long to receive the whole of it, from the ankle to the insertion of the glutæus muscle”.

The man passed the day in a good deal of pain; had frequent vomitings and lost a good deal of blood”. He did well eventually, and “on the seventh day the cavity was lightly filled with dry lint, and a turnip poultice was laid over it because I find in general that the turnip poultice is less uneasy than one of carrots and is remarkably speedy and powerful in correcting the fætor of putrid ulcers and therefore particularly valuable in an hospital.” The patient was discharged with three inches of shortening in June, 1782, eleven months after the operation. He was reported later to have gone back to sea, having “laid aside his crutch, and gotten a strong and useful limb free from pain or swelling.”

Park concludes his letter modestly, saying: “I beg that I may not be so far misunderstood as to have it supposed that the method I have been recommending will certainly succeed in every case. I know the contrary and fear that after the Chirurgic art has done all that it is capable of, many of these diseases will still occur in which Amputation only can preserve the life of the patient. I am conscious that the mode of operating which I have described is by no means perfect but still stands in need of the finishing hand of a more able master.”

For the rest Park married on May 1, 1776, Elizabeth, the eldest daughter of John Ranicar, of West Leigh, Lancashire, and by her had two sons and seven daughters. He was appointed surgeon to the Liverpool Infirmary in 1767, resigned in 1790, continued to practise until 1815, and died at Wavertree on Jan. 22, 1831. From 1769 to 1830 he kept a Register of the midwifery cases he attended, and amongst the 4000 patients perhaps the most momentous was that on “29th December, 1809. A. Gladstone at Rodney St.” She was Anne, daughter of Andrew Robertson, Provost of Dingwall, and the second wife of Sir John Gladstone. The boy was (The Right Honourable) William Ewart Gladstone.

The portrait is from the engraving of a drawing made in 1832 by T. Hargraves and engraved by George T. Doo, kindly lent by Mr. Raymond Tinné Berthon.

THREE CASES OF GLOMANGIOMA OR ANGIONEUROMYOMA (PAINFUL SUBCUTANEOUS TUBERCLE)

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THE name glomangioma has been suggested by Bailey¹ to describe the simple tumour of the neuro-myo-arterial glomus. This rare neoplasm arising from the specialized arteriovenous anastomoses of the skin appears to be a well-defined pathological entity, for not only has it a highly characteristic histological appearance, but in most of the recorded cases it has caused symptoms which are striking and apparently pathognomonic. In fact, as Greig² points out, the symptomatology is so definite that retrospective diagnosis is justifiable and so allows the grouping of cases which have gone under very different names. This nomenclature, varying from 'angiosarcoma' of the German writers to 'painful subcutaneous tubercle' of the English (Wood, 1812, quoted by Greig), is a reminder of the insecure pathology of earlier days. It is probable that some of the so-called peritheliomata have been neoplasms of this type.

The first systematic investigation of the histology and histogenesis of these tumours was carried out by Masson,³ and other workers have confirmed the value of his findings. As a result of his work, interest has been renewed in the structure and function of the normal glomus (Popoff⁴). This is now known to be a much convoluted, modified arteriole communicating directly with a vein. It possesses a characteristic cellular wall, with a rich perivascular nervous network; dilatation and constriction are thought to occur independent of the state of the parent vessel. The narrow channel with its thick coat of cuboidal cells has been called the Sucquet-Hoyer canal. The glomera are most common in the extremities, especially in the nail-beds. The glomus coccygeum, for whose site it is difficult to find a reason, is structurally similar, and presumably serves a comparable purpose. The cutaneous glomus is believed to act as a type of shunt, and to exercise some as yet incompletely understood function in the maintenance of the circulation in peripheral parts exposed to cold, and in the regulation of temperature (Grant and Bland⁵).

Recent references to these tumours are almost entirely American, including a brief annotation by Love,⁶ and reviews by Raisman and Mayer,⁷ Lewis and Geschickter,⁸ Stout,⁹ and Bailey¹ with addition of new cases. In presenting our cases we would refer the reader to these reviews for fuller discussion. The first of the tumours to be described by us was removed by one of us (W.A.M.) in Glasgow. The other two occurred in Australia, and we are very grateful to Dr. Keith Inglis, Lecturer in Pathology, University of Sydney, for histological preparations and clinical notes, and for his permission to publish the cases.

CASE HISTORIES

Case 1.—The patient (M.R. 162/1936), a labourer, 24 years of age, was referred to the Out-patient Department of the Western Infirmary, Glasgow, for treatment of what was believed to be a localized collection of varicose veins on the middle of the front of the left shin. This had been present, to his knowledge, for fourteen years, for the most part without inconvenience. For the last two years, however, he had had increasing pain, which finally led him to consult his doctor. This pain, which he described as a feeling of stiffness and soreness, was situated in the lesion, and radiated laterally to the calf and up and down the front of the leg, and to the knee. It was aggravated by exercise, especially when, at his work, he carried heavy loads. Occasionally pressure over the site caused pain to shoot up the front of the thigh. The lesion appeared to be more sensitive to heat than to cold, and latterly his sleep had thus been much upset. He noted that when painful the lesion became congested, swollen, and dark in colour. There was no history of trauma.

Examination revealed a mulberry-like mass with a coarsely nodular surface, bluish in colour as if engorged with blood; it was $1\frac{1}{2}$ in. in vertical length by 1 in. in breadth. The patient made no complaint of tenderness when it was examined. The first impression was of a localized varicosity, but the absence of change in the remainder of the saphenous system, the long history, and the very slow growth led to a diagnosis of venous angioma.

So thin was the overlying skin that some sacrifice of this at operation seemed probable. Under local anaesthesia, a short transverse flap was therefore raised; the lesion was then seen lying almost free, like a bunch of small grapes, in a pocket in the subcutaneous fat. It was dissected out with ease, and found to be united, not to a vein, but merely to a thin, fairly firm pedicle, which was clamped and ligated. It seems probable that this included the afferent vessel of the tumour.

Case 2.—(S.H. 74/1920.) A man of 59 years, under treatment for epithelioma of the lower lip, showed on the medial aspect of one elbow a pedunculated soft blue mass, which was removed for examination. It is not recorded whether the lesion was painful.

Case 3.—(S.H. 16001/1936.) A boy of 12 years presented a painless swelling on the lateral aspect of the knee, considered to be the result of a blow with a cricket ball seven years before. At operation, an unencapsulated tumour of fatty vascular appearance was found, lying antero-lateral to the tendon of the biceps femoris. It was unattached to skin, or to any of the deeper structures.

HISTOLOGICAL APPEARANCES

Case 1.—The tumour is a roughly circumscribed angioma, with a well-developed stroma and slight condensation of the surrounding tissue. The most distinctive histological feature is the presence, in definite relation to the blood spaces, of large numbers of cuboidal cells. These cells, separated from the endothelium by a basement membrane of varying thickness, are typical glomus cells, as seen in the Sucquet-Hoyer canal of the normal glomus, roughly cuboidal, showing a circular nucleus and a clear and mostly structureless cytoplasm. There is a distinct collagenous limiting membrane round each cell (*Fig. 172*).

The general architecture of the tumour (*Fig. 173*) shows a zonal arrangement, which may be correlated for purposes of description with the distribution of the glomus cells. *In the peripheral zone*, the vessels are of relatively small calibre and appear to be but slightly convoluted; they show a uniform tubular coat of several layers of glomus cells, lying directly under the endothelium. The vessels lie in a fatty areolar connective tissue, occasionally forming a leash, and giving the appearance of a cellular mass traversed by vascular spaces (*Fig. 174*). In one of the vessels in this zone a few muscle fibres are seen between the endothelium and the glomus cells; this conforms to Masson's 'first type vessel', which he presumes to be afferent to the tumour. *Toward the centre of the growth* the arrangement becomes more cavernous, with irregular blood spaces lined by endothelium, which is separated from the supporting fibrous framework by only two or three layers of glomus cells. *In the central parts of the tumour* the stroma shows thickening into nodules of fibromyxomatous nature with some myxomatous degeneration. The intervening vascular clefts show a prominent

endothelium but no definitely related glomus cells, although collections of these are scattered throughout the fibromyomatous tissue. Many are morphologically identical with the glomus cells in the rest of the tumour, but all forms of transition are seen between the glomus cells

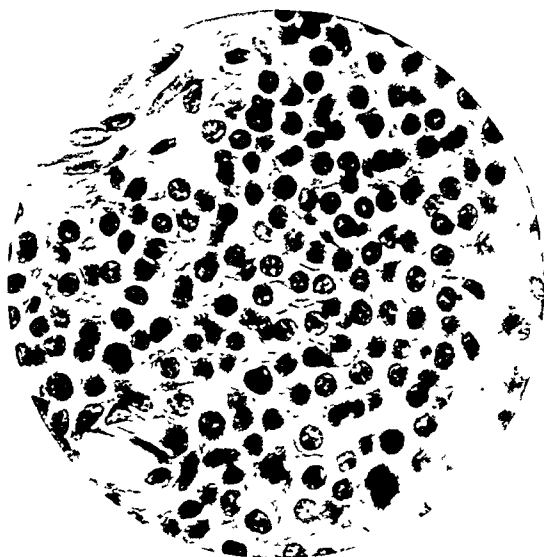


FIG. 172.—*Case 1.* This shows part of a perivascular sheet of typical glomus cells. The cells are uniform in size, cuboidal in shape, with rounded nuclei rich in chromatin, and show a distinct limiting membrane. ($\times 500$.)



FIG. 173.—*Case 1.* General structure. The main tumour mass is cavernous in arrangement, with areas of fibromyomatous proliferation of the stroma. The lowest part of the field shows the periphery of the tumour. ($\times 15$.)

and the indisputable smooth muscle cells present in the nodular areas (*Fig. 175*). Such appearances lend support to the hypothesis that glomus cells are essentially modified muscular cells; to these Masson applies the epithet 'myoid'. Some of the glomus cells show in their

cytoplasm the minute fuchsinophil granules and twigs which Masson considers in his material to be the precursors of smooth muscle fibrils. No mitoses are seen.

The peripheral regions of the growth show small myelinated nerve-trunks, while in the central area there are numerous groups of single myelinated fibres scattered about in the



FIG. 174.—Case 1. Peripheral zone. The peripheral type of vessel, with its thick tunic of glomus cells, is seen below. Above, the blood spaces are larger and irregular, and the glomus cells are fewer. ($\times 50$.)



FIG. 175.—Case 1. This field shows a less orderly arrangement of vessels and glomus cells, the latter forming an almost continuous sheet. Note the gradual transition between typical cuboidal glomus cells and fusiform cells. ($\times 200$.)

fibromyomatous stroma. The presence of non-myelinated fibres in our tissue, embedded as it is in paraffin, cannot be demonstrated to our satisfaction. No Pacinian corpuscles have been seen in the sections examined.

Elastic tissue (*Fig. 176*) is irregularly scattered throughout the stroma of the growth, showing no constant relationship to the glomus cells. In places it is finely distributed among them with a condensation either internal or external to the cell mass; elsewhere no fibres



FIG 176—*Case 1*. Central zone. This shows the elastica irregularly proliferated and with no constant relationship to the blood spaces. (*Weigert's elastica stain*) ($\times 80$)



FIG 177—*Case 3*. This is from the central part of the tumour, and shows the small vascular spaces lying in a wide sheet of glomus cells. In parts, the glomus cells are dissociated by œdematous swelling of the stroma. ($\times 150$)

are seen among the glomus cells, but a distinct lamina separates them from the surrounding connective tissue. In the nodular areas of the stroma the elastica shows both the sharply stained coils characteristic of proliferation, and the flat appearance and fainter staining of degenerative change

Case 2.—The histology of this case is so nearly identical with that of Case 1 that a separate description is unnecessary. The two lesions may be considered in essence as simple tumours in which all the elements of the normal glomus participate; in many parts there is a fairly accurate reproduction of the highly evolved structures characteristic of the parent organ.

Case 3.—The appearances in this case are, by contrast, less 'organoid', the proliferation being mainly of glomus cells, with but little demarcation of the tumour into zones. The general picture (*Fig. 177*) is of smallish vascular spaces lying in broad sheets of glomus cells. The spaces are lined by a prominent endothelium supported in most places by a layer of fibrillar tissue with rather indeterminate elongated cells. These seem to show transition to the surrounding glomus cells. Between the perivascular masses the glomus cells are less closely packed, and lie separated from each other in a loose oedematous ground-work. Areas are seen here also with cells apparently transitional between glomus and smooth muscle cells, but actual muscle elements are few and mainly lying loose; there is none of the comparatively firm fibromyomatous tissue seen in the other cases. No nervous elements are noted.

DISCUSSION

The main points of clinical interest in these tumours are the sites of origin, the pain, which may be extreme, and the occasionally associated autonomic upset.

Glomangioma may arise presumably wherever there are normal glomera. These are most abundant on the extremities, especially in the nail-bed and in the pulp. Of the 65 cases collected by Bailey, the tumours were on the hand in 30, and 20 of these were subungual. Four of Stout's 11 cases were subungual on the hand. The distribution on the leg shows no special distal localization, half of the 18 reported tumours having been on the thigh.

The pain which is such a characteristic feature of the majority of these tumours, especially when subungual, is described variously by the patients as bursting, stretching, or burning. It tends to originate in the tumour and to radiate over an increasing area, till in the end it may affect an entire limb or even more. It is frequently brought on by pressure or even a touch, so that the patient may develop a protective mannerism—for example, to prevent the clothes from touching the lesion; in other cases, changes of temperature elicit the spasms of pain. During the paroxysm, engorgement of the tumour has often been noted. On the other hand, the patient may fail to realize that the unobtrusive nodule is the source of his pain. The tumour may be present for years before pain makes its appearance, as in our *Case 1*; while in the case reported by Picard¹⁰ and in our *Case 3*, the tumour was definitely painless. It is likely that tumours arising in the restricted tissue of the nail-bed bed will earlier cause pain than those in soft parts; this may account for the more frequent recognition of tumours in this site.

In one of the cases described by Masson (Barré's case), a subungual glomangioma on the left middle finger appeared to have given rise to disturbance of the autonomic nervous system, for the affected arm was warmer than the other, and there was a homolateral Horner's syndrome. These phenomena disappeared after excision of the tumour.

Bailey states that nearly half the recorded cases give a history of a single severe trauma to the site. So far as is known, it is unusual for trauma to play so important a part in the etiology of other simple neoplasms.

The clinical diagnosis of the typical case is easy, and depends principally on recalling the existence of these tumours. Many have been regarded as ordinary angiomas, of capillary or venous type, or as neurofibromata. In making a

differential diagnosis, one should consider also the various simple subungual growths, and, in addition, melanoma, myoma cutis, synovioma, rheumatic nodule, and painful cicatrix. On the other hand, the histological appearances have occasionally in the past been misinterpreted; the cellular nature of the growth leading even to the unfortunate diagnosis of sarcoma.

The treatment of most of the recorded cases was removal of the tumour under local anæsthesia; this led almost invariably to cure. Stout reports the second example of local recurrence. In a few instances pain persisted for a short period after operation.

The position is most aptly summarized by Picard's epigram: "Auch hier gilt, wie bei allen seltenen Erkrankungen als erste Bedingung, das Darandenken—die zweite, die Behandlung ist einfach: Exstirpation." (In dealing with this, as with all rare lesions, the most difficult step is to bring it to mind—this done, its treatment is simple: extirpation.)

The photomicrographs are by Mr. John Kirkpatrick, of the Department of Pathology, University of Glasgow.

REFERENCES

- ¹ BAILEY, O. T., *Amer. Jour. Pathol.*, 1935, xi, 915.
- ² GREIG, D. M., *Edin. Med. Jour.*, 1928, xxxv, 565.
- ³ MASSON, P., *Lyon chir.*, 1924, xxi, 257.
- ⁴ POPOFF, N. W., *Arch. of Pathol.*, 1934, xviii, 295.
- ⁵ GRANT, R. T., and BLAND, E. F., *Heart*, 1931, xv, 385.
- ⁶ LOVE, J. G., *Proc. Staff Meet. Mayo Clin.*, 1935, x, 593.
- ⁷ RAISMAN, V., and MAYER, L., *Arch. of Surg.*, 1935, xxx, 911.
- ⁸ LEWIS, D., and GESCHICKTER, C. F., *Jour. Amer. Med. Assoc.*, 1935, cv, 775.
- ⁹ STOUT, A. P., *Amer. Jour. Cancer*, 1935, xxiv, 255.
- ¹⁰ PICARD, H., *Zentralb. f. Chir.*, 1931, lviii, 2133.

THE PROBLEM OF ANURIA

A REVIEW OF RECENT WORK ON RENAL PHYSIOLOGY, WITH REPORTS OF TWO CASES

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THE cause and nature of anuria have been debated, at times hotly, for over a century, yet we are still little nearer to a solution than when Chopart in 1821 described a case of calculous anuria in which the post-mortem showed that the unobstructed kidney was apparently normal. It is noteworthy that this early case is an example of the most interesting and most puzzling type of anuria, which will be referred to in this paper as 'reflex anuria'. This term is chosen, not in any way to beg the question of the reflex nature of the condition, but for lack of any other term in general use. If I may define it more exactly, I would say that by it I mean a complete cessation of urinary output from the kidneys following an obstruction of one ureter in a subject whose other, unobstructed, kidney is capable of function.

There are two methods of approach to the problem, the clinical and the experimental, and both present peculiar difficulties which have prevented any certain, or indeed even strongly presumptive, conclusion from being reached. On the clinical side, the rarity of the cases, their rapid onset as emergencies in cases which have not as a rule been previously investigated, the difficulty of full investigation in patients so gravely ill, and the difficulty of interpreting post-mortem findings in terms of living physiology and pathology, combined with our ignorance of many of the details of the physiology and pathology of the kidney and of the true nature and cause of the intoxication produced by suppression of urine, all make the problem a very difficult one. The most striking and puzzling feature of reflex anuria is its rarity compared with the frequency of its apparent exciting cause. It is much to be regretted that case records have been notably lacking in certain important details which might have indicated a possible path towards a solution, or at least have prevented fruitless search along certain other paths. It may be that future cases carefully recorded will prove more helpful.

With regard to the experimental method, the difficulties are scarcely less formidable. So complicated is the structure of the kidney, and so numerous the factors concerned in the production of urine, that many aspects of its physiology are still uncertain. Nervous and hormonal influences, blood-pressure, the composition of the blood, its viscosity and osmotic pressure, temperature, urine pressure, are only some of the factors which have to be controlled and taken into account. Moreover, it is doubtful how far it is justifiable to apply knowledge gained by experiments on isolated and often denervated kidneys to the function of the kidneys in the living body. Again, different kidneys, even when isolated, show the same inexplicable and apparently capricious differences of behaviour under the influence

of apparently identical factors which are shown by kidneys in the living body. Lastly, the production of anuria by stimuli simulating those presumed to be active in cases of reflex anuria in human beings is exceedingly difficult. It was apparently achieved in a few cases by Goetzl¹ many years ago, but subsequent workers have failed to obtain his results.

Nevertheless, in spite of these difficulties in the way of a direct attack on the problem of anuria, fresh knowledge of the physiology of the kidney has gradually been gained by physiologists. So far this fresh knowledge does not appear to have been applied to the problem of anuria. The time has not yet come when it is possible to bridge the gap, but a short review of the recent physiological work may point the way to a more direct attack on the problem.

Types of Anuria.—Anuria is classified by Thomson-Walker² as :—

1. Circulatory (severe depression of blood-pressure).
2. Reflex.
3. Infective.
4. Obstructive.

Swift Joly's³ classification is :—

1. Pre-renal (low blood-pressure).
2. Renal (infective and toxic).
3. Post-renal (obstructive).

It is the 'reflex' type on which the chief interest centres.

Historical.—The question round which controversy has raged ever since Chopart's⁴ case in 1821 is this : Assuming that a reno-renal or uretero-renal reflex exists, is it capable of producing a complete suppression of the function of a normal kidney, or only of a kidney already rendered more or less inefficient by disease ? Legueu⁵ and Morris⁶ held the view that though reflex anuria might occur, it only did so when the unobstructed kidney was diseased, never when it was perfectly healthy. More recently Winsbury-White⁷ and Kelly and Birnam⁸ have also expressed this opinion. Thomson-Walker² holds the same view, though he admits that a few cases have been recorded in which the unobstructed kidney was *apparently* healthy, though not proved to be so. On the other hand, Albarran,⁹ Young and Davies,¹⁰ Rovsing,¹¹ Watson,¹² and many others have expressed the opinion that reflex anuria might occur when the unobstructed kidney was normal. The difficulties in the way of a solution of this question are emphasized by a statement of Young's¹⁰ that he has been unable to find any record of uræmic death due to reflex anuria where the unobstructed kidney could be shown to be healthy. He concluded, not that such cases of anuria do not exist, but that they always recover. Even should death occur, the unobstructed kidney would be found greatly congested (this point is further discussed below) as a result of the immediate condition, and this would of itself make it impossible to estimate small degrees of abnormality which might have been present before the anuria commenced. The conclusion seems to be that reflex anuria may occur in cases in which the unobstructed kidney has been shown to be so near normality as to make the question one of academic interest only.

Swift Joly³ has recently raised a much more important and fundamental question. He states that in all cases of so-called reflex anuria there is a severe renal colic, and suggests that the suppression of urine in the unobstructed kidney is capable of being explained by the marked fall in the general blood-pressure caused by the

severe painful spasm of the obstructed ureter. There are, however, strong arguments against this theory. The pressure in the renal pelvis is nil, and filtration of urine should take place as long as the filtration pressure (that is, the pressure in the glomeruli) exceeds the osmotic pressure of the colloids in the blood-plasma. This may be taken as 25 mm. of mercury; the glomerular pressure is given as two-thirds of that in the renal artery; secretion of urine should therefore only cease when the blood-pressure falls below 45 mm. of mercury. In dogs and cats, whose blood-pressure is only slightly below that of a human being, it has been found that secretion of urine ceases when the blood-pressure falls below 30 to 40 mm. of mercury; other authorities give 75 mm. of mercury as the minimum blood-pressure in dogs at which secretion is possible. In any case it seems unlikely that the pressure would be sufficiently lowered by renal colic to produce a complete suppression of urine. It is unfortunate that, so far as I have been able to find, in none of the records of cases of reflex anuria has the blood-pressure been stated.

There is another important clinical observation which bears on the problem. Those who have seen the unobstructed kidney in cases of reflex anuria (it has been exposed on several occasions either deliberately or as the result of a mistaken diagnosis of the side obstructed) agree that it is enlarged and deeply congested (Rubritius,¹³ Albarran⁹). Too little stress has been laid on this observation. It has been assumed by the advocates of a reflex action that anuria is brought about by a 'vascular cramp' of the kidney, but as Swift Joly points out, if this were the case, one would expect to find a small, pale, bloodless kidney. Frank,¹⁴ reviving a suggestion of Israel's, postulates a "congestive reflex" in the unobstructed kidney, but his theory is stated in such vague terms as to be of little value, and he adds what may be the dangerous conclusion that if for any reason operation is not possible the correct treatment would be to lower the blood-pressure by purgation and fluid restriction.

CASE REPORTS

The following cases are recorded because of certain unusual features which they present, and of a new therapeutic measure employed in one of them.

Case 1.—E. C., a married woman of 60, very stout and rather 'wheezy', attended the Casualty Department of the Middlesex Hospital on July 21, 1935, with an attack of severe pain in the right side of the abdomen. The pain came on suddenly and was followed by retching and vomiting. It was situated all down the right side of the abdomen, and was not referred to the back or groin. The patient had had two similar attacks in the past two years, but they had not been so severe. She had never been jaundiced. She was referred to the Out-Patient Department, where I saw her the next day, July 22. The pain had then passed off. There was no abnormality of micturition, the bowels were open regularly. She had had rheumatic fever at the ages of eighteen and twenty-four. She had had eight children. Recently she had been treated for blood-pressure.

ON EXAMINATION.—She had dyspnoea on exertion. There was no jaundice. The tongue was clean, and she had a high-pressure pulse. There was an ill-defined bulging of the abdominal contents between widely divaricated rectus muscles. There was tenderness on pressure over the right side of the abdomen, maximal over the gall-bladder. I agreed with the diagnosis of gall-stone colic which had been made in the Casualty Department and ordered a cholecystogram. Three days later I received a message asking me to see the patient, who was in the X-ray Department having the last of her cholecystograms taken. I found that she had passed urine on the evening of July 22, the day I had seen her, three days before, but had passed none since. On examination she appeared, and said she felt, quite well. The

tongue was clean and moist; there was no œdema. She had had no more pain. The bladder was catheterized and found to be empty. Cystoscopy was performed; the bladder was very intolerant and held only two ounces. There was a red œdematous swelling in the position of the right ureteric orifice, which was not itself seen. The left ureteric orifice had a normal appearance, but no efflux was seen. She was admitted under the care of Mr. (now Sir) Alfred Webb-Johnson, and radiographs of the urinary tract were immediately taken.

RADIOGRAPHY.—The cholecystograms which had just been completed showed a normally filling and emptying gall-bladder, and no calculi either in the urinary or in the bile passages. The right kidney shadow was, however, extraordinarily distinct, in spite of the obesity of the patient; there was no visible shadow of the left kidney. Radiographs of the pelvis were not very clear on account of the patient's obesity, and on account of the shadow in the colon of unabsorbed opacol.

July 26.—The day after admission and the fourth day of anuria she was drowsy and vomited several times. She was a little puffy under the eyes; there was no œdema of the ankles and no twitching. She passed $2\frac{1}{2}$ oz. of urine at 7 a.m. The blood-urea was 250 mg. per cent. A hot-air bath caused her to sweat profusely. At 3 p.m. she was catheterized and the bladder found empty. The blood-pressure was 180 systolic. At 5 p.m. continuous intravenous infusion of normal saline by the drip method was commenced, and at midnight she was given 10 c.c. of 20 per cent saline by the same route; the total amount of saline was 4 pints.

July 27.—In the early hours of the morning she passed 11 oz. of urine, and during the twenty-four hours the total passed amounted to 74 oz.

July 28.—She passed a small oval grey smooth stone. There had been no more pain. From this time she had been passing satisfactory quantities of urine. Up to this time the temperature had been raised occasionally to 99° , and the pulse had been between 64 and 72.

July 29.—Blood-urea 180 mg. per cent. On close questioning she gave a history of a single attack of left-sided pain about Christmas, 1934.

July 31.—Blood-pressure 170/80. Urine: specific gravity 1010, alkaline reaction, contained albumin. The urine continued to show albumin until her discharge.

Aug. 6.—Blood-pressure 150/60. Blood-urea, 77 mg. per cent. Microscopical examination of the urine showed triple phosphate crystals, no casts.

Aug. 10.—Radiographed. Preliminary film showed no visible shadow of either kidney. Five minutes after intravenous injection of 20 c.c. of uroselectan B the pelvis and calices of the right kidney were visualized. The left kidney showed no secretion of uroselectan throughout the investigation.

I was anxious to perform a chromo-cystoscopy so as to establish the functioning or non-functioning of the left kidney, but the patient resolutely refused any further investigations, and discharged herself on Aug. 14.

Points of Interest.—

1. The mistaken diagnosis.
2. The failure of the calculus to show in the radiographs.
3. The exceptional density of the shadow of the right kidney in the radiographs taken during the period of anuria.
4. The unusual signs and symptoms of anuria.
5. The recovery of a severe case of anuria without operation.

Discussion.—The case unfortunately throws no light on the problem of reflex anuria. So far as the evidence went, the obstructed right kidney was the sole functioning organ; it is possible that the function of the left kidney had been destroyed by impaction of a stone—invisible on the radiographs, or since passed without her knowledge—which had caused left renal colic at Christmas. The very clearly defined shadow of the obstructed kidney is of great interest, and has not, to my knowledge, been previously noted. It is presumably due to the congestion of the kidney, and in certain cases in which there is uncertainty as to the side obstructed, might help the surgeon to decide on which side to perform a

nephrostomy. In cases of reflex anuria, however, if it is true that both kidneys are greatly congested, one would expect to see a dense kidney shadow on both sides in a radiograph. Even so the observation would be of value in showing that both kidneys were capable of function. The anuria was unusual in its symptoms, in that the period of tolerance was short (three and a half days) and there was puffiness of the eyelids. The presence of a previous chronic nephritis is suggested by the history of pre-existing high blood-pressure and the persistence of albuminuria for sixteen days after urinary secretion was re-established; no casts were found, however. The patient was a bad operative risk, and the conservative treatment was justified by its result in this case, though not to be recommended in a case of equal severity in a subject of better operative risk. Secretion was only re-established after relief of the obstruction by passage of the stone into the bladder; but it would be tempting Providence too much to rely on this happy event taking place at the critical moment in every case.

Case 2.—K. D., a girl of 15, was admitted to the West London Hospital under the care of Mr. G. F. G. Batchelor, on June 10, 1932. She complained of attacks of severe pain in the right groin, of a gripping nature, without definite radiation; lasting six or seven hours; occurring about once a week; and accompanied by vomiting. They had been going on for about eighteen months. Micturition was unaffected. Radiography of the urinary tract showed slight distortion of the calices on the right side. Examination of a catheter specimen of urine on June 18 showed no pus and a few *Staph. albus*. Cystoscopy on June 28 showed a normal bladder, and ureteric orifices from which clear effluxes were seen. The ureters were catheterized, and specimens from both sides showed no pus and were sterile on culture. The urea content was, on the right, 1.0 per cent, and on the left 1.5 per cent.

July 5.—*Operation.* The right kidney was exposed and found to be very low and extremely mobile; the pelvis was slightly dilated. Nephropexy was performed by Thomson-Walker's method and the wound closed except for a tube drain.

July 8.—She was well and passing water freely. The temperature remained a little raised (maximum 100°), but settled to normal on the twelfth post-operative day. She was passing from 40 to 80 oz. of urine per day.

July 20.—She was allowed to get up.

July 21.—She complained of some pain in the left side. Passed 30 oz. of urine.

July 22.—She vomited several times during the night, and had two convulsive attacks during the morning; these consisted of colonic and tonic stages; she bit her tongue in one attack. She passed no urine after midnight, and catheterization revealed an empty bladder. The blood-pressure was 180/120. The blood-urea was 82 mg. per cent. The right kidney was palpable.

July 23.—The convulsions continued and she was unconscious. She was still secreting no urine. The temperature shot up to 106°; and the pulse to 160. She was given 30 oz. of 4 per cent glucose intravenously. A spinal anæsthetic of 0.4 cg. of stovaine was given. Cystoscopy was performed, and both ureters catheterized, and a flow of urine commenced. The catheters were left in, and drained 40 oz. of urine during the next twelve hours. There were no more convulsions, and improvement was rapid. The temperature remained swinging up to 101° and 102° for another ten days, there was pus in the urine, and the right kidney was palpable and tender.

Aug. 12.—The blood-urea was 30 mg. per cent. On Sept. 9 she was discharged.

Points of Interest.—

1. Both kidneys were proved to be healthy before operation.
2. The anuria seems to have been due to the effect of an acute and virulent infection, possibly with an added element of obstruction by a clot (which was seen in the bladder on cystoscopy). It is impossible, however, to place the case definitely in one category or the other.

3. The symptoms seem to have been due to a sudden intense toxæmia due to an acute infection, and not to uræmia.

4. The blood-pressure was raised.

5. A form of treatment—spinal anæsthesia—not to my knowledge previously described, was used.

Discussion.—It occurred to me after considering the teaching of the late Mr. Tyrrel-Gray on the part played by a sympathetic reflex in ileus, and the effect which a spinal anæsthetic sometimes has in that condition of causing a spontaneous action of the bowels, that if, in this case, there was obstruction of one ureter by a blood-clot, and a consequent reflex effect on the other kidney, a spinal anæsthetic should break the reflex arc and enable the unobstructed kidney to resume its secretion. Mr. Batchelor, under whose care the case was, kindly allowed this suggestion to be carried out. Since, in this case, on account of the desperate condition of the patient, three different therapeutic measures were all employed in rapid succession, it is impossible to attribute the successful result which was obtained to any one of them separately. I believe, however, that in a case of reflex anuria a spinal anæsthetic should be a valuable addition to our therapeutic armamentarium. In order to be effective it should on theoretical grounds produce anæsthesia reaching as high as the sixth dorsal segment. Even a much lowered blood-pressure would be no contra-indication, for in shock due to peripheral-nerve stimulation a spinal anæsthetic raises the blood-pressure by cutting off the stimuli which have caused it to be depressed (O'Shaughnessy and Slome¹⁵).

In this connection Neuwirt's case¹⁶ quoted by Swift Joly is of interest. In a case of severe unilateral renal colic, unrelieved by morphia, with extreme oliguria (330 c.c. in twenty-four hours), he injected novocain round the semilunar ganglia on both sides. The pain was relieved in fifteen minutes and 2000 c.c. of urine were excreted in the next twenty-four hours. Cystoscopy next day showed that both kidneys were working. Unfortunately Neuwirt does not record the blood-pressure, so that this interesting case, which might have settled one of the problems of reflex anuria, leaves it still unsolved. This method of posterior splanchnic block has the disadvantages that it is not without danger, and that it requires an experience which few, at any rate in this country, possess. The method of spinal anæsthesia is almost free from these disadvantages, and it is advanced as having a possible value in certain cases of reflex anuria, before recourse to the more drastic method of nephrostomy.

ANATOMY

Nerve-supply.—The kidney is supplied by nerve-fibres from both the sympathetic and the vagus, which run to the hilum on the surface of the vessels of the pedicle. Those from the sympathetic are derived mainly from the 6th dorsal to the 1st lumbar segments of the cord, though possibly some fibres come from as high as the 4th dorsal segment. There is no experimental indication of the function of the vagus, nor is there evidence of any direct effect of either vagus or sympathetic on the secretory cells.

Blood-supply.—The vascular system of the kidney is peculiar. The arterioles from the vasa recta break up into a capillary plexus in the glomeruli; these capillaries reunite into a second set of arterioles, which again break up into a second capillary

plexus around the convoluted tubules. The second capillary plexus drains into the venous radicles. Hence the capillaries of the glomeruli lie between two sets of arterioles capable of contraction and relaxation.

RECENT WORK ON THE PHYSIOLOGY OF THE KIDNEYS

There is evidence that the two sets of arterioles—afferent and efferent in relation to the glomerular capillaries—are independent and different in their response both to nerve stimulation and to drugs and hormones, and that by their contraction and relaxation they control the glomerular pressure which is the most important single factor in determining the output of urine. The capillaries themselves have not been shown to have any active contractile power.

We may briefly summarize the work which has elucidated the behaviour of these vessels under different conditions. Most of this work has been done by perfusing kidneys with different concentrations of adrenalin. Low concentrations and high concentrations of adrenalin have different effects, and it appears that this is due to a difference in the reactions of the afferent and efferent arterioles.

Richards and Plant¹⁷ showed that in low concentrations adrenalin caused an increase in the kidney volume and an increased flow of urine, and suggested that it did so by constricting the efferent vessels and thus increasing the glomerular blood-pressure. Later, in collaboration with Schmidt,¹⁸ Richards confirmed his theory by direct microscopical observation of the glomerular capillary tufts in the kidneys of frogs, perfused with different concentrations of adrenalin. With low concentrations, the number of patent glomeruli increased, and the glomerular tuft enlarged; it became more densely congested with blood-cells, and the rate of flow of the cells through it was slowed. They concluded that this was due to constriction of the efferent arterioles. With higher concentrations there was a diminution of the number of patent glomeruli, of the lumen of the individual capillary, and of the number of patent capillaries in any one glomerulus; these effects they concluded were due to constriction of the afferent arterioles. It is also of interest to note that a decrease in the number of patent glomeruli is caused by stimulation of a lower dorsal sympathetic ramus, of the sciatic nerve, and by large doses of pituitrin, while increase in the number of patent glomeruli is caused by section of the lower dorsal sympathetic rami, by anoxæmia, by the diuretics sodium chloride (isotonic), glucose, urea, and caffeine, and by pituitrin in small amounts. The effectiveness of this mechanism is strikingly illustrated by an observation of Hayman and Starr¹⁹ that, whereas under the influence of caffeine all the glomeruli were patent, high concentrations of adrenalin might cause closure of all but 10 per cent.

Winton²⁰ carried the investigation a stage further by perfusing isolated heart-lung-kidney preparations with defibrinated blood containing varying concentrations of adrenalin, and recording the blood-flow from the renal vein and the urine-flow from the ureter. With weak concentrations he found that in the majority there was increased flow of urine and decreased flow of blood. As a result of mathematical deductions as to the glomerular blood-pressure, he concluded that the effect on the arterioles was (1) dilatation of the afferents, and (2) constriction of the efferents. There were, however, two kidneys out of seventeen which behaved anomalously and showed a diminished flow of urine and a diminished blood-flow. With higher concentrations of adrenalin he found a diminished flow of urine and a diminished

blood-flow. He concluded that both the afferent and the efferent arterioles were constricted. He makes another interesting observation that the effect of adrenalin was antagonized by ergotoxin.

The effect of nerve section and nerve stimulation has not been worked out in such detail, but in general is as follows (Fee²¹):—

1. Section of the nerves of the renal pedicle—increased blood-flow, increased kidney volume, increased flow of urine.

2. Weak stimulation of the nerves of the pedicle—increase in volume of kidney and in flow of urine.

3. Strong stimulation of the nerves of the pedicle—decrease in kidney volume and in flow of urine.

Thus it appears likely that the afferent and efferent arterioles respond to nerve stimulation in the same way as to adrenalin.

In this work there are several points which may have some bearing on the problem of anuria.

1. The important role played by the efferent arterioles in the control of glomerular pressure and the flow of blood through the glomeruli.

2. The independence of action and difference in reaction both to drugs and to nervous stimuli shown by the afferent and the efferent arterioles.

3. There appears to be a balance between the two systems of arterioles which varies in different kidneys. One is reminded of sympathetic-parasympathetic balance, which varies in different species, in different individuals of the same species, and in different emotional, reflex, and metabolic states in the same individual. The difference of response of different kidneys to caffeine supports this conclusion. In all cases caffeine causes an increase in blood-flow through the kidney; the diuretic effect is less constant—sometimes it is marked, sometimes absent. The explanation given by Verney and Winton²² is that, while in most cases the dilatation of the afferent vessels is greater than that of the efferent vessels, causing a rise in glomerular pressure, in those cases in which diuresis fails to occur, this is not the case, but both dilate equally. This inconstancy of behaviour marks all experimental work on kidneys. On the clinical side it suggests an analogy with the rarity of reflex anuria compared with the frequency of its apparent exciting cause.

The Anuria of Exercise.—It has been shown²³ that anuria occurs during severe muscular exercise in spite of a marked rise of blood-pressure.

The Influence of Hormones.—Theobald and Verney²⁴, working with kidneys which had been completely denervated, caused inhibition of water diuresis by afferent-nerve stimulation. They advance reasons for thinking that the effect is produced on the arterioles by a hormone liberated by nerve stimulation. They believe that this hormone is not adrenalin, but may be post-pituitrin, which is well-known to have a similar inhibiting effect on water diureses. The effect is not due to lowering of blood-pressure. They suggest that the anuria of exercise may also be due to a hormone.

Reabsorption in the Tubules.—Duncan Morison²⁵ states that in hydro-nephrosis the contents of the renal pelvis are constantly being changed. If glomerular filtration is still going on there must be some process of re-absorption. He investigated the nature of this re-absorption by introducing various dyes and Indian ink into the pelvis, and obstructing the ureter. He found that there are

two routes of re-absorption, by the lymphatics and by the tubules. If foreign substances, and even particulate substances such as Indian ink, can be absorbed by the cells of the convoluted tubules, it would appear probable that not only water and threshold substances, as usually believed, but non-threshold substances also—in fact, the entire glomerular filtrate—can be re-absorbed when there is obstruction to the outflow from the tubules. There are other experimental indications that this may be the case.

The Effect of Ureter Pressure.—If the ureter pressure is gradually raised, there is at first no decrease in the flow of urine, but if it is progressively raised beyond a certain point, usually about 10 mm. of mercury, the output of urine falls, and finally ceases when the ureter pressure reaches 20 or 30 mm. of mercury.

The Effect of Venous Obstruction.—Partial venous obstruction produces a congested kidney with a diminished blood-flow and diminished output of urine. The engorged venules press upon the urinary tubules and obstruct them, thereby raising the urine pressure (Winton²⁶). As with the ureter pressure, there is a critical pressure below which an increased venous pressure has no effect on the flow of urine. This critical point is the same for both venous pressure and ureter pressure. Moreover, in partial venous obstruction, increase of ureter pressure has no effect on the urine flow unless the ureter pressure exceeds the venous pressure.

These observations suggest that in the normal unobstructed kidney there is a certain intrarenal pressure acting in all directions within the capsule of the organ, and tending to obliterate the tubules, thus offering a certain constant resistance to the production of urine. The raised intrarenal pressure associated with œdema or engorgement may be an important factor in producing the oliguria which occurs in certain diseased conditions of the kidneys (Winton²⁷). It seems to me that this conception of intrarenal pressure may be the key to the problem of anuria.

The Factors which Influence the Output of Urine.—From the above considerations, it is now possible to obtain a fairly clear picture of the factors determining the output of urine. They are:—

1. *The Urine Pressure.*—This is normally nil, but may rise as a result of partial or complete obstruction of either the ureter or the tubules. Obliterative pressure on the tubules is one of the effects of venous obstruction.

2. *Changes in the Secretion-reabsorption Activity of the Tubule Cells.*—We still lack precise knowledge of this activity, but it would appear probable that it depends, like that of other secretory processes, on the blood-supply of the cells. There are also indications, previously discussed, that it is modified by the urine pressure.

3. *The Blood-pressure in the Glomerular Capillaries.*—This pressure, less the osmotic pressure of the serum proteins and the urine pressure, is the effective filtration pressure. It is increased by: (a) Rise in general blood-pressure; (b) Relaxation of the afferent arterioles; (c) Contraction of the efferent arterioles.

4. *The Area of the Capillary Bed from which Filtration is taking Place.*—This depends on the state of contraction or relaxation of the afferent and efferent arterioles, and on general blood-pressure. In a kidney under normal conditions only a small fraction of the glomerular capillaries is open to blood-flow; the number is greatly increased during diuresis, and under other conditions it may be diminished.

5. *The Osmotic Pressure of the Colloids in the Plasma of the Blood Contained in the Glomerular Capillaries.*—It is probable that if any change occurs it is of so slight a degree as to be negligible in its effect on urine filtration.

Causes of Anuria.—If we apply this conception of the production of urine, it is evident that there are several possible causes of anuria.

1. *Urine Pressure.*—Increased urine pressure caused by obstruction of the ureter or the tubules. Obstruction of the tubules is a result of venous obstruction, and we may assume that it is also a result of a rise in intrarenal pressure due to oedema of the renal parenchyma.

2. *Arterial.*—(a) Marked fall of general blood-pressure; (b) Contraction of afferent arterioles.

3. *Venous.*—Obstruction of veins.

Hypothesis as to the Nature and Causation of Reflex Anuria.—Bearing in mind that the evidence is in favour of a congested kidney in reflex anuria, it is possible that the sequence of events may be thus: Spasm of one ureter in response to impaction of a stone sets up afferent nervous impulses which, either by direct reflex action or by release of some hormone substance, cause vascular changes in the other kidney, an alteration in capillary permeability, and an exudation of fluid into the renal parenchyma; the raised intrarenal pressure obstructs the tubules and raises the urine pressure opposing filtration; at the same time it causes partial obstruction to the venous outflow so that the kidney is congested with blood. Contributory factors may be: (1) Lowering of general blood-pressure; (2) Abnormal leak of urine back through the tubules into the blood-stream, as a result of increased urine pressure.

Such a tentative hypothesis, while it is of no value in itself without proof, may be of some value in suggesting lines of attack on the subject. It brings reflex anuria into some relation with toxic anuria (in which the kidneys are congested and oedematous, and have on occasion been restored to function by release of intrarenal pressure by decapsulation), and with those cases of anuria following urethral or ureteral instrumentation which are variously classified as toxic and reflex.

It is possible of course that reflex anuria may be of varied types, and that in some cases the anuria may be due to a reflex vascular spasm.

Another interesting possibility is suggested by recent work on pyeloscopy. It has been shown that the calices and pelvis have a regular cycle of diastole and systole, and that during the diastole of a calix the connecting channel between calix and pelvis is completely shut. Spasm of the circular muscles of these channels or of other portions of the upper urinary tract may be caused reflexly by disease of the gall-bladder, appendix, or viscera having an associated nerve-supply, and may disappear when the diseased organ has been dealt with (Jona,^{28, 29}). This suggests the possibility that the suppression of urine from the unobstructed kidney in reflex anuria might be due to an intense reflex spasm at the pelvi-ureteric junction or the necks of the calices.

The value of ureteric catheterization is now well recognized; it may be that it drains urine pent up above such a spasm. It was suggested many years ago by Essex Wynter, though never published by him.

SUMMARY

1. The nature of the problem of reflex anuria is stated, and a brief review of the history of the controversy is given. The conclusions reached are, firstly, that the unobstructed kidney which is the subject of reflex anuria may be free from gross

disease, and secondly, that the 'vascular cramp' theory of reflex anuria fails to account for the observed fact that the unobstructed kidney is swollen and congested. There are certain objections to the theory that the anuria is due solely to lowering of blood-pressure. It is pointed out that a record of the blood-pressure in all cases of reflex anuria would help to solve the problem in its clinical aspect.

2. Two cases of anuria are reported. While neither of these can be certainly put in the class of reflex anuria, they both present unusual and interesting features. In the first case the obstructed kidney showed as a very dense shadow in the radiograph, presumably on account of its congestion. The contralateral kidney was probably functionless. The ureteric calculus which was subsequently passed did not show in the radiograph. The symptoms of anuria were early in their appearance. Restoration of the flow of urine, probably due to passage into the bladder of the stone blocking the ureter of a solitary functional kidney, followed the administration of intravenous saline.

In the second case complete anuria followed nephropexy; both kidneys had previously been proved to be functioning perfectly. The cause was probably partly obstructive and partly infective. The symptoms were unusually early and severe. Restoration of the flow of urine and relief of symptoms followed a combination of intravenous glucose, a spinal anæsthetic, and bilateral ureteric catheterization.

Spinal anæsthesia is put forward as a rational therapeutic measure worthy of trial in cases of reflex anuria. This applies whatever its etiology, since the afferent path at least must be a nervous one.

3. There has been a considerable amount of experimental work done by physiologists in the last few years dealing with the blood-supply of the kidneys, its response to humoral and nervous influences, and the effect on the secretion of urine of these changes in blood-supply, and of changes in the urine pressure. This work has an important bearing on the problem of anuria, and does not seem to have been noticed in the surgical literature. Winton's conception of intrarenal pressure in particular is full of possibilities, and requires further work. A short summary of this recent physiological work is given, and a tentative explanation of reflex anuria is suggested.

I wish to express my thanks to Sir Alfred Webb-Johnson and to Mr. G. F. Grant Batchelor for permission to report the cases under their care, and for their encouragement and helpful suggestions and criticism, and to Dr. F. R. Winton, whose fine work has led to a new conception of renal physiology, for giving me the benefit of his expert knowledge and criticism.

REFERENCES

- ¹ GOETZL, A., *Pflüger's Archiv. f. d. ges. Physiol.*, lxxxiii, 128.
- ² THOMSON-WALKER, SIR J., *Urological Surgery*, 1914.
- ³ SWIFT JOLY, J., *Stone and Calculous Disease of the Urinary Organs*, 1929. Heinemann.
- ⁴ CHOPART, quoted by Eliot, H., *Ann. d. Mal. d. Org. gén.-urin.*, 1911, i, 107.
- ⁵ LEGUEU, P., *Clinique*, Paris, 1914, ix, 482.
- ⁶ MORRIS, SIR H., *Diseases of the Kidneys and Ureters*.
- ⁷ WINSBURY-WHITE, *Stone in the Urinary Tract*, 1929. Churchill.
- ⁸ KELLY and BIRNAM, *Diseases of the Kidneys, Ureters and Bladder*, 1922, ii, 112.
- ⁹ ALBARRAN, *Congrès d'Urologie*, 1898, quoted by Eliot, H., *Bull. Soc. anat.*, 1909, June, 353.

- ¹⁰ YOUNG, H. H., and DAVIS, D.M. *The Practice of Urology*, 1926, i, 385. Saunders.
- ¹¹ ROVSING, *Zeits. f. urol. Chir.*, xii, Nos. 3 and 4.
- ¹² WATSON, J., *Amer. Jour. Med. Sci.*, 1909, cxxxvii, 550.
- ¹³ RUBRITIUS, H., "Die Klinik u. Therapie d. Anurie", *Verh. d. Deut. Gesells. f. Urol.*, VII Kongress, 1926, 233.
- ¹⁴ FRANK, L., *Surg. Gynecol. and Obst.*, 1915, xx, 526.
- ¹⁵ O'SHAUGNESSY and SLOME, *Brit. Jour. Surg.*, 1935, xxii, 589.
- ¹⁶ NEUWIRT, *Zeits. f. urol. Chir.*, xi, Nos. 3 and 4.
- ¹⁷ RICHARDS and PLANT, *Amer. Jour. Physiol.*, 1922, lix, 144.
- ¹⁸ RICHARDS and SCHMIDT, *Ibid.*, 1927, lxxi, 178.
- ¹⁹ HAYMAN and STARR, *Jour. Exper. Med.*, 1925, xlii, 641.
- ²⁰ WINTON, F. R., *Jour. of Physiol.*, 1931, lxxiii, 151.
- ²¹ FEE, *Ibid.*, 1929, lxviii, 305.
- ²² VERNEY and WINTON, *Ibid.*, 1930, lxix, 153.
- ²³ MACKEITH, PEMBREY, SPURRELL, WARNER, and WESTLAKE, *Proc. Roy. Soc. (B)*, xcv, 413.
- ²⁴ THEOBALD and VERNEY, *Jour. of Physiol.*, 1935, lxxxiii, 341.
- ²⁵ DUNCAN MORISON, *Proc. Roy. Soc. Med.*, 1928, xxii, 219.
- ²⁶ WINTON, F. R., *Jour. of Physiol.*, 1931, lxxii, 49.
- ²⁷ WINTON, F. R., *Proc. Physiol. Soc.*, 1933, Feb. 18; *Jour. of Physiol.*, lxxvii.
- ²⁸ JONA, J. L., *Surg. Gynecol. and Obst.*, 1933, lvi, 632.
- ²⁹ JONA, J. L., *Ibid.*, 1934, lix, 713.

SOME CONTRIBUTIONS TO THE CAUSATION, PATHOLOGY, AND TREATMENT OF DUODENAL ULCER AND ITS COMPLICATIONS

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THE etiology of gastric and duodenal ulceration, together with certain aspects of its prognosis under varieties of treatment, medical and surgical, is still the subject of controversy throughout the world. The literature abounds with accounts of the relative merits of medical and surgical treatment, and the Continental surgeons vie with their British colleagues in arguing the case for radical as against conservative surgery. Meanwhile, thousands are suffering from this disease with imperfect treatment of one sort and another; and more recently the advent of histidine has raised the hopes of many, although observers in some hospitals have found it to be uncertain in its results.

The literature of duodenal ulceration is extensive, and there would be little excuse for adding to it, unless one had some definite contribution to make towards the solution of the problem of its treatment. The series of cases whose treatment and observation forms the subject of this paper is a large one, consisting of 2500 consecutive cases, all treated at the same clinic; and all treated surgically, though by various methods, either by or under the personal supervision of the authors of the paper. While this clinic is in India, a country with special conditions which influence the course of disease, and with a village population the nature of which prevents in many cases a satisfactory follow-up of patients, it is hoped and believed that the findings from these cases will prove to be of value in the search for truth which it is incumbent upon all surgeons to prosecute. If it can help to decide what is still a vexed question—namely, what is really the best treatment for duodenal ulcer, and whether that treatment should vary according to the condition of the ulcer, or should be standardized—it will have achieved its object. It is hoped, therefore, that lessons learned in India may afford a definite contribution to the knowledge in possession of surgeons in other parts of the world.

In South India, so-called peptic ulcer is very common—six hundred times as common as in the north. Probably five in every thousand of the adult population of Travancore suffer from duodenal ulcer; gastric ulcer is not nearly so frequent an occurrence. On one occasion, one of us (T. H. S.) operated on five brothers, all of whom were suffering from ulcer of the duodenum. But even in the State of Travancore, comprising the southern 200 miles of the West Coast of India, the distribution of peptic ulcer is not uniform. In the extreme south of the State it is comparatively rare; on the sea-coast it is less common than inland; and in the northern, and especially the central, half of the country, it is very prevalent indeed.

Dr. S. H. Pugh, at Neyyoor Hospital, S. Travancore, was the first surgeon

to perform gastro-jejunostomy for duodenal ulcer in South India; this he did against the advice of his medical colleagues, who protested against surgical treatment for dyspepsia. A similar experience befell Major Bradfield in Madras (now Surgeon-General) a few years later. But both these surgeons justified themselves, and to-day surgeons of the Madras General Hospital are operating on hundreds of these cases every year, while in Travancore over 600 operations for duodenal ulcer are performed annually.

The observations in this paper are based on an experience of 2500 cases of duodenal ulcer, operated upon by various methods during the last ten years at the Neyyoor Hospital. While the follow-up of many of these cases is of necessity imperfect, so many of the patients having been poor and uneducated villagers, yet there are few hospitals where such a large continuous series of cases is available; and more than 600 cases have been followed up and personally examined.

PHYSIOLOGY

The stomach when considered physiologically is usually regarded as divided into four portions (*see Fig. 181*):—

1. The cardiac portion, the glands (tubuloracemose) of which secrete mucus; oxyntic cells are frequent, and a certain amount of the acid secretion of the stomach is done here.

2. The fundus, in which there are (a) short tubular glands lined with 'central' cells which appear granular and are said to secrete pepsin, (b) lateral (oxyntic) cells which have a prolongation of the tubule into them, and secrete acid. The major portion of the acid is secreted in the fundus.

3. The 'gastric pathway' or small curvature and pre-pyloric portion. Here there are no oxyntic cells; the mucous membrane is less mobile; lymphoid nodules are numerous; and mucin is secreted abundantly. The glands are long and tubular, and are formed of cells of one kind only, supposed to secrete pepsin, and probably also secreting an acid-producing hormone.

4. The pyloric antrum, containing cells which secrete mucus and long tubular glands secreting pepsin. The secretions from the pyloric antrum are said to produce the anti-anæmic factor; and anæmia of the microcytic hypochromic type is a recognized complication of operations such as Polya's gastrectomy in which the pyloric end of the stomach has been removed, and is not a common complication of gastrectomies in which the pyloric antrum is left, the middle portion of the stomach being removed. Conflicting reports have been made of this question of anæmia, and Morley¹⁵ states that anæmia is rare after Schoemaker's operation, in which it is just this pyloric portion that is removed. Gordon-Taylor¹⁶ reported a high rate of anæmia—44 per cent of cases—after gastrectomy, while Lake¹⁷ in a similar series found anæmia to be very rare.

The results of our operations seem to show that if a portion of the pyloric antrum is left, gastrectomy is not followed by secondary ulceration of the gastro-jejunal type, and this important result makes it worth while preserving the pyloric antrum whenever it is possible to do so. Further research is required as to the question of anæmia; but it is certain that Addisonian anæmia is rare after gastrectomy, and the usual microcytic type can be so easily relieved by the administration of ferrous salts that it is not a very serious consideration.

PATHOLOGY

The duodenal ulcers of South-west India show certain atypical characteristics ; nevertheless the proportion of gastro-jejunal ulceration after gastro-enterostomy in our series of cases is exactly the same as it is in similar series collected elsewhere (see especially Garnett Wright's series reported in the *British Journal of Surgery*, 1935, xxii, 433). This would indicate that the lesion is essentially the same as it is in other countries ; but the fact remains that there are certain features of our cases which are by no means typical.

The most striking feature is the rarity of perforation ; in our series of 2500 ulcers, only 4 cases of perforation were seen. Hæmorrhage also is very rare. These exceptional features are consistent with the known and very striking tendency of the Indian abdomen to chronic rather than acute ailments. Chronic appendicitis

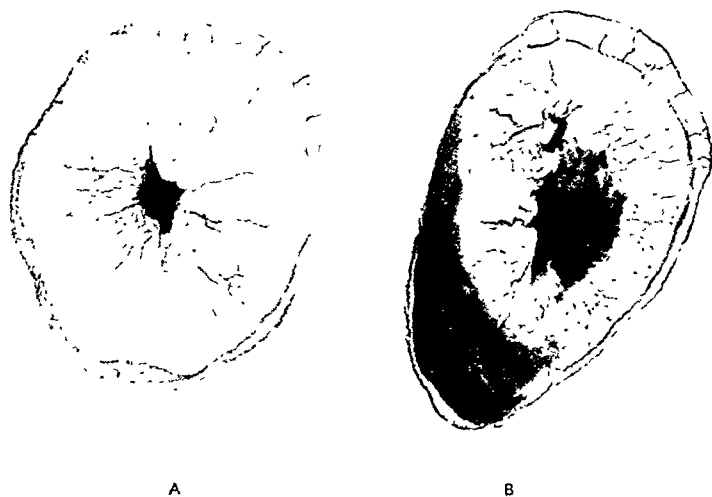


FIG. 178.—Pylorus from two cases of early duodenal ulcer. A, Three months' history—slight duodenitis ; B, Eight months' history—marked duodenitis with small healed ulcer. (Natural Size.)

is very common, and acute appendicitis is rare ; only 20 cases were recorded at Neyyoor last year out of 3000 major operations, whereas over 200 cases of chronic appendicitis were operated upon. Similarly, duodenal ulcer tends to be chronic rather than acute ; and, together with the rarity of perforation, we have noticed the extreme frequency of large cicatrized masses of scar tissue, with stenosis of the duodenum and a tremendously dilated stomach. We see this condition in over 40 per cent of all ulcers of the duodenum or pylorus in South India.

As far as the histology of the stomach is concerned, there is a great prevalence of changes in the mucosa at a distance from the ulcers, obviously indicating a common pathological change throughout a great part of the stomach, of which the ulcer itself is but a local exaggeration. In the earliest stages (Fig. 178) there is

found at the pylorus, especially on the duodenal side thereof, a congestion of the mucosa which indicates a duodenitis, and which clinically is associated with pyloric spasm, hunger pain, gastric hyperacidity, and in fact is indistinguishable from a case of definite duodenal ulceration.

In several of these cases multiple small superficial ulcers in the pyloric region have been found at the time of operation, but they are of early occurrence and apparently heal up in a few months as the simple duodenal ulcer develops. At a considerable distance from the pylorus there is an increase in the size and frequency of lymphoid nodules and the mucous membrane shows an aggregation of lymphocytes; occasionally this is very dense and continuous throughout the gastric mucosa, which is (with the entire submucosa) densely infiltrated with round cells. The glands of the mucosa are distorted, the tubules short and filled with cells. *Lymph nodules in the worst cases showed areas of necrosis in their centres, suggestive of small abscesses; possibly the breaking down of one or more of the lymphoid follicles in this way is the actual determining factor in starting an ulcer, and is almost certainly the process by which the multiple ulcers are formed.*

Thus it appears that a chronic gastritis and/or duodenitis is the precursor of ulcer. This finding is in accordance with that of Aschner and Grossmann, of New York, Konjetzny,¹ Puhl, etc.² (See also Carswell,³ Faber⁴; Hurst and Stewart are of different opinion, but on negative grounds—*Gastric and Duodenal Ulcer*, p. 56.)

ETIOLOGY

The various theories put forward in recent books upon the subject will be summed up as headings only:—

1. *The ulcer diathesis*,⁵ said to be of two kinds, gastric and duodenal.⁶
2. *The acid factor*, said to be a cause of ulceration by Bolton⁷ and Hurst,⁸ but a result of it by other writers.
3. *The neurogenic factor* (Cushing, Rokitanski), sometimes doubtless a predisposing cause.
4. *The toxic factor* (Rosenow,⁹ Wilkie¹⁰).
5. *The embolic factor*, of doubtful significance.
6. *The dietetic factor*.

The Dietetic Factor.—Much has been written about the first five factors, but the dietetic factor has received little attention, and in a recent massive work is dismissed in three lines as an “accessory factor”. It is undeniable, however, that diet has a very large influence on the incidence of duodenal ulcer, and is more than an accessory as far as South India is concerned. The distribution of duodenal ulcer in Travancore (*Fig. 179*), where the part of that country with the densest population (the southern one-third) is that which has the lowest number of cases of ulcer, seems to point to a dietetic cause, as does the fact that the disease is far commoner in those parts of India where tapioca is eaten. It is an interesting fact that the Tinnevely district of S. India, which used to be almost free from duodenal ulcer, has recently adopted the habit of eating tapioca-root, and is now producing a continually increasing number of cases of ulcer (*Fig. 180*).

Starting in 1927, Sir Robert McCarrison¹¹ made experiments on rats, feeding them on diets obtained from Madras and Travancore respectively.

1. Of rats fed for 675 days on Madrassi diet—11·1 per cent developed gastric ulcer.

2. Of rats fed for 675 days on Travancore diet—27·7 per cent developed gastric ulcer and 11·1 per cent developed a severe duodenitis.

3. Controls, well-fed rats—no cases of ulcer.

The ulcers in the rats which were fed on the Madrassi diet were in the synomous or proximal portion of the stomach; those with the Travancore diet developed ulcers in the distal or mucous portion of the stomach.

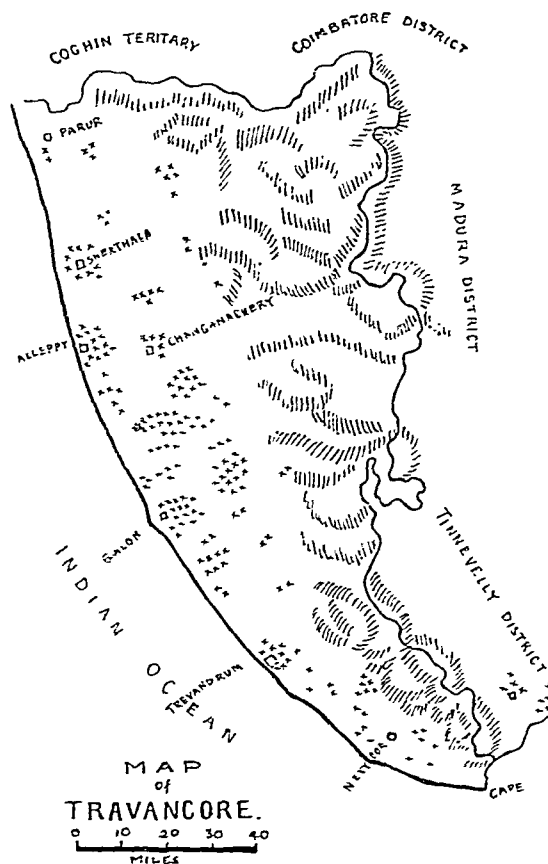


FIG. 179.—Map of Travancore, showing Neyyoor in the south; the crosses represent the areas from which peptic ulcer cases come.

This is in conformity with our clinical experience that gastric ulcer is relatively far more common in the Madras district than it is in Travancore, where duodenal ulcer outnumbers the gastric variety by 30 to 1.

In the rats fed with Travancore diet (tapioca, rice, chillies, pepper, and a little fish) the stomachs were dilated, the mucous membrane frequently congested, especially in the region of the pylorus, and some cases had shallow ulcers in this situation. The duodenum was often markedly congested, and there was

round-celled infiltration of many of the glandular organs, with occasional small hæmorrhages. The interesting thing about these observations is the fact that they correspond closely with the microscopic appearances we have seen in patients suffering from duodenal ulcer in Travancore. One is forced to the conclusion that the high incidence of duodenal ulcer in Travancore is due to the diet of the country, especially of the poorer classes, and that the ulcer itself is but a local manifestation of a general change in the tissues brought about by dietetic factors.¹²

What is the dietetic factor at fault, and how does it produce its effects?

a. The acid factor. The acidity, as obtained from fractional test-meals, of the South Indian, whether suffering from gastric or duodenal ulcer or not, is certainly slightly higher than what is considered normal in the west, except when he has gastritis, in which case acid is often low, and mucus content very high.

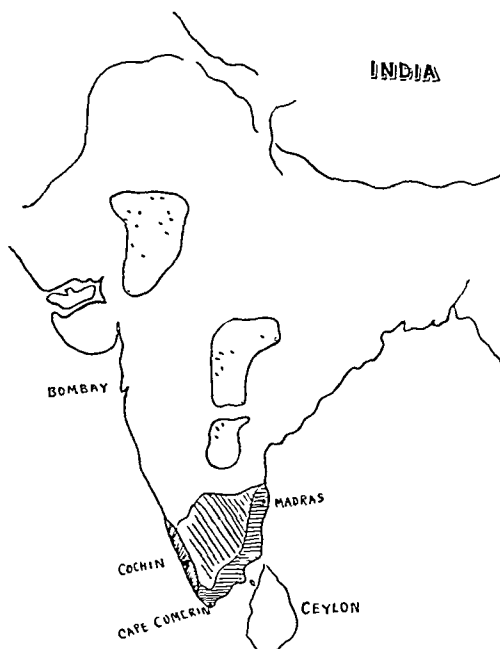


FIG 180.—Map of India showing incidence of peptic ulcer. The deeply shaded portion represents Travancore and Cochin and South Malabar, where the diet is mainly tapioca root and rice, and the medium shading represents the Madura and Tinnevely districts and East Madras Presidency, where rice is the staple food supplemented by gram and other grains. In this area peptic ulcer is common, but not as common as in Travancore. The oblique shading represents the remainder of Madras Presidency, where the diet is more varied and peptic ulcer less common. The stippled patches represent the wheat-growing areas, where duodenal ulcer is very rare.

b. The nervous factor and *c. The embolic factor* need not be considered in a country where half the time of most people is spent in watching the rice grow, and where such ulcers as do develop practically never bleed.

d. Trauma may, however, play some part. The highly spiced hot food, containing fibrous stringy vegetables, must inflict on even the most robust of stomachs some measure of trauma. But the general irritating nature of the South Indian diet is much the same in various parts, although the incidence of duodenal and gastric ulcer varies so much from one part to another; so we have to look further than trauma for the real effect of the diet as an ulcer-producing agent.

e. Vitamin deficiency. The great majority of our ulcer patients live on tapioca and rice, an almost exclusive carbohydrate diet with but little vitamin. Meat and fish are rarely taken; milk, eggs, and most fruits are luxuries used only by the well-to-do, who are not typically the class of case with peptic ulcer. Vitamins A, B, and C are especially deficient in the Travancore poor man's diet; and it has already been mentioned that this community has duodenal ulcer at a rate of incidence six hundred times greater than is the case in the Northern Punjab, where the people are said to have one of the best balanced diets in the world. In Travancore itself, peptic ulcer is infinitely more common in the central part of the State, where tapioca and rice form the staple diet, than in the southern portion, where a larger variety of vegetables, with rice and little or no tapioca, is eaten. One of us (I. M. O.) investigated the records of the State Troops of Travancore, who live in barracks with a well-controlled diet. In the last five years there have been only two (unproved) cases of duodenal ulcer among them; yet these same men come from villages where there is an incidence of duodenal ulcer of something like 1 per cent of the population. (In a hospital on the sea-coast, where we act as consulting surgeons, it has been noted that all their peptic ulcer cases come from the coolie population working in the fields inland; ulcers are very uncommon amongst the fisher folk who live by the sea and get a lot of fresh fish to eat.)

The function of vitamin A is "to maintain the functional integrity of the cells covering body surfaces, thereby preventing invasion of the organism by microbic agents" (McCarrison).

"One of the first demonstrable changes in avitaminosis is an alteration in the distribution of bacterial life within the lumen of the intestinal tract. The bacteria ingested by the mouth are not destroyed in the usual manner, but remain viable for many hours. . . . There is a gradual and progressive loss of the power of the body surface to control microbic life" (Lloyd Arnald).

These two extracts are sufficient to show that the high incidence of duodenal ulcer in Travancore is very likely due to vitamin—especially vitamin A—deficiency. This, by lowering the defences of the gastric mucosa, leads to an invasion of it by bacteria, and a chronic gastritis and duodenitis. The rupture of small abscesses in the lymph-follicles leads to ulcer formation, and the ulcers thus formed, if continually irritated by a highly spiced diet, refuse to heal but become chronic. This would seem to be the etiology of South India; and it is at least possible that vitamin deficiency is responsible for far more gastric and duodenal ulceration than has hitherto been supposed.

Chronic appendicitis—probably having a similar etiology—is very often associated with the duodenal ulceration; in nearly every case we operate upon in Neyyoor appendectomy is performed as a routine procedure; 73 per cent of all cases show some tenderness in the appendix region at the time of clinical examination.

EXAMINATION OF PATIENTS

The usual and obvious points, of history, etc., will not be mentioned. We now make the following examinations as a routine, not so much for diagnostic purposes as to indicate the line of treatment.

Test-meal.—Very high acidity, especially if this is maintained for some time, is an indication for gastrectomy of the Finsterer type; a large portion of the acid-secreting area of the stomach being thus removed.

Ordinary high acidity may be met with in a case in which the pylorus is stenosed, and in that case is no indication for gastrectomy as against gastro-jejunostomy, for we have found that a case of stenosis of the pylorus is best dealt with by gastro-jejunostomy. A low acidity may mean in some cases a chronic gastritis, of significance from its indication of a rigorous medical treatment during convalescence; a very low acidity (especially if coupled with a loss of appetite) being characteristic of malignancy.

X-ray Examination.—This is best carried out by the surgeon himself with a fluorescent screen, as the combination of vision with palpation gives him more information than a series of photographs taken by another person, except in certain cases in which the visibility of a crater and its persistence in successive radiographs are of importance for the diagnosis.

1. The size, position, and mobility of the stomach, and the activity of its peristaltic waves, may all be noticed, together with any irregularities it may possess.

2. The filling of the duodenal cap and its irregularities can be compared with any tenderness that is noted. At the same time the patient can be turned round, and notches in the shadow of the stomach or duodenum not visible in the antero-posterior position may be seen, and compared with local tenderness.

3. The time of emptying is important as a deciding factor in determining what operation should be done. Putting it broadly, we have found that the type of duodenal ulcer which is definitely tender on palpation but which is associated with rapid emptying of the stomach is best treated by a gastrectomy of the Finsterer type, especially if it is also associated with hyperchlorhydria; if treated by any other surgical method a jejunal or gastro-jejunal ulcer is very likely to be the sequel. If, however, there is delay in emptying the stomach (especially if that delay, with a liquid barium meal, is over six hours), the case will probably do well with a gastro-jejunostomy, unless acidity, as found by the fractional test meal, is very high. ↘

TREATMENT

Under many circumstances, peptic ulcer is a medical condition. In early cases, and where circumstances permit, a course of medical treatment is indicated before resort to surgical measures.

But for the Indian villager—and nearly all of the cases considered in this article are of that type—medical treatment is out of the question, or was until the recent days of histidine. Certain articles, such as tapioca or chillies, can be forbidden; but there is no guarantee that he will ever obtain the articles necessary to balance his diet, or that he will submit to a rigid régime when he is once outside the walls of a hospital. To most Indian villagers, food means curry and rice, and the problem before us is to fit his stomach for the curry and rice which he will almost certainly put into it when he goes out of the hospital. It has therefore come about that in our clinic at Neyyoor we have almost universally resorted to surgery in the treatment of gastric and duodenal ulcer. ↘

GASTRO-ENTEROSTOMY

At first, gastro-jejunostomy was our routine procedure, but as time goes on we have seen, in common with surgeons elsewhere (though often independently of them), that there are definite indications for other operations. Throughout our

experience, however, gastro-enterostomy has remained the operation of choice for cases where there is definite stenosis of the pylorus, with a healed or quiescent duodenal ulcer, and without any exceptionally marked hyperchlorhydria.

CAUSES OF FAILURE AFTER GASTRO-ENTEROSTOMY

Out of the two thousand and odd cases in which gastro-enterostomy has been done by us during the last ten years, we have had two main classes of failure.

The first class is those in which the *pylorus was not stenosed*; the ulcer was therefore not short-circuited, and food (often of an irritating nature) continued to pass the pylorus. In 5 per cent of all cases followed up, the persistence of the duodenal ulcer, and its irritation by food and gastric juice, led to continued pain and symptoms.

In these cases, we carried out a resection of the pyloric antrum—and sometimes of the duodenal ulcer itself—and thus converted the gastro-enterostomy into a Billroth II. The results of these cases were often very striking, a system of one-way traffic being thus introduced, and allowing the duodenal ulcer, if it was not excised, to heal. For some years we found that this type of secondary operation was very satisfactory, and in 1934 one of us (T. H. S.) reported that “a recurrence, or gastro-jejunal ulcer, has never been observed” in these cases. Since then, however, we have had to operate for gastro-jejunal ulcer on two patients who had previously had operations of the Billroth II type.* Out of 63 cases of this order, we have to report these two cases of gastro-jejunal ulcer, although the first 56 cases had been free from ulceration after a secondary Billroth II operation. The remedy for this class of failure is to perform a suitable gastrectomy for cases of duodenal ulcer with permeable pylorus, especially if there is also high acidity.

The second class of failure is seen in cases who are *indiscreet in their diet* at an early stage. It is this category which includes most of the cases of gastro-jejunal ulceration. Some of the less civilized of our patients have been known to escape from the hospital seven or ten days after their operation, and, going to a coffee-shop, to order a meal of curry and rice. Several times we have seen extremely early development of gastro-jejunal ulcer—in one case (No. 2300) in eleven days after the primary operation a well-marked ulcer was found at the anastomosis.

Other causes of trouble after gastro-enterostomy are as follows, and each of them bears an indication for the careful performance of the operation in the right way, rather than for the performance of some other operation instead.

1. Use of improper suture material. A certain Government Hospital in South India was in the habit, some years ago, of using a silk suture for the gastric mucosa; several cases whose gastro-enterostomy had been performed there came to us later with symptoms of gastro-jejunal ulcer. Of these, two were found to have ulceration (Nos. 1736 and 1809 in *Table III*); the remainder, some six or seven cases, had no ulcers, and recovered completely after we had removed the silk thread through a small incision in the stomach.

2. In a few cases gastro-jejunal ulcer was due apparently to a stoma placed

* Note.—Of the two cases of ulcer recurring after Billroth II operation, one was done by T. H. S. at Neyyoor; the other was done elsewhere. From our own cases we have only the one recurrence to note.

improperly. The stoma must reach the greater curvature, or it may allow an accumulation of food in the stomach in front of it, with consequent dragging on the stoma itself owing to the weight of the imperfectly emptied stomach.

3. If there is literally 'no loop' on the proximal end of the jejunum, the stomach may be held up in an unnatural position and the anastomosis dragged upon, with a resulting predisposition to ulcer in its neighbourhood.

4. Performance of gastro-enterostomy in a case without duodenal ulcer, but perhaps with chronic gastritis or high acidity, is inexcusable, and is only mentioned here for the sake of completeness, as being a cause of trouble later.

5. The stoma may be too large or too small. We have found the best average to be two and a half inches; if the stomach is very dilated, the stoma should be longer, as it will contract when the stomach contracts.

6. The loop for a gastro-enterostomy should not, as is well-known, be too long, or regurgitant vomiting may occur; it is best about two to three inches, but may be far longer in a Polya gastrectomy.

7. We have had two cases of recurrent ulcer owing to the gastro-enterostomy having been performed (at other hospitals) the wrong way round. Such a thing is by no means unknown in Britain.

COMPLICATIONS OF GASTRO-ENTEROSTOMY

Acute Complications.—These are so well-known that we only wish to record here the routine treatments which we have found effective.

1. *Acute Dilatation of the Stomach.*—This is always heralded by a fairly sudden rise in pulse-rate. Any patient whose pulse rises rapidly has a stomach-tube passed at once, and the stomach washed out. In severe cases, the patient can be turned on his face and the tube left in position for some time. In addition to relieving acute dilatation, the stomach-tube may reveal a hæmorrhage or an excess of bile in the stomach, either of which may cause distress and a rise in pulse-rate.

2. *Hæmorrhage.*—The stomach is washed out, and 30 min. of adrenalin solution in an ounce of water is run in. Morphia and hæmoplastin are injected, and the patient laid flat.

All our cases of hæmorrhage in recent years have responded well to this treatment, and the only ones who have died (two) are those who have had an immediate second operation to stop the hæmorrhage. If such an operation is performed, the hæmorrhage is usually not seen, having stopped at the time of operation; and if the patient does not die as a result of the second operation, he may bleed again when reaction sets in. If transfusion is to do good, it must be done before the patient is too weak. It is usually unnecessary in these cases. If the stoma is made parallel to the main vessels in the stomach (i.e., transverse to its long axis) and if, when the mesocolon is sewn to the stomach wall the principal vessels in the neighbourhood are included in the five or six interrupted stitches required, fatal bleeding will hardly ever occur.

3. *Lung Complications.*—These are often avoided by giving pneumococcus immunogen *before* operation in all cases whose chest is not absolutely sound. If pneumonia develops, full doses of ammon. carb. and ipecac. are surprisingly well tolerated by a recently operated stomach, and save many patients from severe lung complications. Carbon dioxide inhalations soon after operation are a good prophylactic.

4. *Vomiting*.—Vomiting, besides that which is associated with dilatation of the stomach, is sometimes due to the unwonted *presence of bile in the stomach*, which has been for a long time unused to its presence owing to pyloric obstruction. Many of these cases are relieved by a stomach-tube and wash-out, followed by the administration of an acid mixture (15 min. of dilute sulphuric or hydrochloric acid in 2 oz. of water) from time to time. Sodium bicarbonate makes them worse, for it is the alkaline bile to which the stomach objects. Frequent drinks of water are almost invariably good, and the total withholding of fluids so often advocated is often very harmful.

If the vomiting is due to a *kink in the small intestine*, as is occasionally the case, a secondary operation may have to be performed, but this should not be undertaken unless it is clear that there is a definite obstruction—something more, that is, than a mere dilatation of the stomach. The vomiting of more than 3 pints of fluid in twenty-four hours is generally considered as an indication for secondary operation; the experienced surgeon will generally be guided by his clinical instinct to see that the vomiting is due to a definite obstruction, either from a kink or at the anastomosis.

Whether this obstruction is due to a kink in the small intestine or to œdema of the anastomosis, the treatment is the same—pass a nasal stomach-tube, and guide it beyond the obstructed place. Fluids, and the vomited gastric contents, can be introduced through this as required. Only if the original operation was technically wrong (e.g., long loops requiring entero-anastomosis) will more operative interference than the guiding of this tube be required.

Table I.—RESULTS IN 635 DUODENAL ULCER CASES FOLLOWED-UP AND PERSONALLY EXAMINED, 1925-35

LENGTH OF TIME AFTER OPERATION	EXCELLENT RESULT	GOOD RESULT	PAIN AS COMPLICATION	INCAPACITATED BY PAIN	APPENDICULAR PAIN OR ADHESIONS	TOTAL
5-10 years	19	7	5	1	—	32
2-5 years	44	3	14	6	—	67
1-2 years	24	5	6	2	—	37
Under 1 year*	24	5	15	10	—	54
Unclassified	345	42	24	28†	6	445
Totals	456	62	64	47	6	635
Percentages	71.5	10	10	7.5	1	100

* Notice the high proportion of painful complications during the first year, indicating that many of these cases settle down and cure themselves, some doubtless with alkalis or diet.

† 16 proved, 12 suspected.

Late Complications.—

1. *Adhesions causing Obstruction.*

2. *Intussusception of Small Intestine through the Anastomosis.*—These are rare complications, the possibility of which must be borne in mind, and the treatment for which is operation. In 2500 cases, we have had one case of intussusception and several of adhesions; but both are very rare, and by far the commonest complications are (3) and (4).

3. *Recurrence of Pain, and Non-healing of Original Duodenal Ulcer.*—This has already been dealt with under the heading of 'Treatment', and is nearly always satisfactorily treated by the simple operation of removing a segment of the stomach and converting the original operation into a Billroth II. A portion of the pyloric antrum should be left, as the mucin it secretes is apparently of beneficial effect in preventing subsequent ulceration. If, however, recurrent ulceration of the gastro-jejunal order does occur, which it did in one of our 63 cases, conditions will still allow a more radical resection of the stomach of the Finsterer type to be performed.

4. *Gastro-jejunal Ulcer.*—In 445 cases, all followed-up by a personal examination of the patients, this complication occurred in 28, of which 16 were proved by operation to have gastro-jejunal ulcer, and in 12 it was strongly suspected. Though various observers have reported percentages varying from one to twenty,¹³ this figure of 6 per cent is about the average, and exactly agrees with Garnett Wright's estimate.¹⁴

In our own series of 2500 cases, we have dealt with 35 of our own gastro-enterostomy cases for gastro-jejunal ulceration. But some of our cases have no doubt been operated on elsewhere, and we, on the other hand, have operated on 46 cases whose first operation was done in other hospitals. Eighty cases of gastro-jejunal ulcer among 2500 of gastro-enterostomy is a percentage of only 3; whether the true figure is 3 or 6, in any case gastro-jejunal ulceration is the most common and the most feared of all complications of gastro-enterostomy.

ETIOLOGY OF GASTRO-JEJUNAL ULCER

Excluding such cases as are due to technical errors in the operation, mentioned above, gastro-jejunal ulceration is always due to a persistence of the conditions which caused the original ulceration. The consideration of the etiology is largely the consideration of the etiology of duodenal ulcer, which has already been done, and the principal factors in its production are:—

1. *Acidity.*—Cases with very high hyperchlorhydria are always prone to develop recurrent ulceration, some of them at a very early date. The fact that gastro-jejunal ulcer has never been seen after operations for gastric carcinoma (G. Wright¹⁴) is doubtless due simply to the fact that in carcinoma acidity is always low, and not to the explanations given by Wright in the paper quoted. I have seen several cases of undoubted gastro-jejunal ulceration cure themselves by frequent drinks of sodium bicarbonate over a long period. There is, therefore, little doubt that the principal cause of the ulcer is acidity, though it is not the only cause, for, in the Association of Surgeons' statistics (G. Wright, op. cit., p. 444) the acidity was found to be low in 20 per cent of cases, and high in only 60 per cent.

2. *Infective Foci.*—These are probably of minor importance, though if they happened to predispose to the original ulcer, and persist, they may also predispose to the recurrent ulceration.

3. *Diet.*—Cases have already been mentioned in which a gastro-jejunal ulcer has been produced within a few days of operation by an injudicious meal; in this case an irritation factor in the diet—and perhaps a straining of the anastomosis itself due to over-filling the stomach—is the immediate cause of the ulcer. But in cases in which a serious vitamin deficiency exists and was the cause of the original ulcer, owing to the considerations already mentioned near the beginning of this paper, the continuance of those same conditions and of the vitamin-deficient diet will always

predispose to a recurrence of ulceration; the natural defences of the gastric and intestinal mucosa are below par, owing to the deficiency of vitamins (especially A).

"The true etiology is unlikely to be known until we have solved that of duodenal and gastric ulcer. Whatever that cause, it is probably still active in the case of secondary ulcer" (*Brit. Jour. Surg.*, xxii, p. 444). In South India at any rate, this question of vitamin deficiency seems to be a fundamental cause of duodenal, and therefore also of gastro-jejunal, ulceration. It is our belief, however, that acidity is the most important of these three causes.

PREVENTION OF GASTRO-JEJUNAL ULCER

The prevention of gastro-jejunal ulcer is threefold:—

1. *A suitable operation* must be done in the first place. Gastro-enterostomy in cases of high acidity with no pyloric stenosis is an unsuitable operation.

2. In other cases, in which gastro-enterostomy is suitable, *it must be done properly*, with the lower end of the anastomosis near the greater curvature of the stomach, and with catgut suture in both layers. The loop should be 2 or 3 in. long, and the anastomosis itself about 3 in. except in very dilated stomachs. No knots should be inside the gut, as is usually advised in text-books, but should always be outside the serous coat. No suture should pass through the interval between stomach and intestine, or infection will be drained from the lumen of the gut into the space where healing is to take place. All stitches involving the mucosa should definitely pass through all coats of the stomach, the suture being started and finished *outside* the organ. So-called redundant mucosa should *not* be removed, as it helps to protect the seromuscular layers from the action of gastric juice. The chief blood-vessels around the anastomosis should be tied by the stitches fixing the mesocolon to the stomach, which should never be within half an inch of the anastomosis itself.

3. *Adequate post-operative treatment* by alkalis and suitable diet must be observed. If recurrence of the ulcer is staved off for a few months after the operation, it is far less likely to occur. Moreover, undoubtedly a few definite gastro-jejunal ulcers heal up with medical treatment.

If all these points are observed, gastro-jejunal ulcer, as well as other complications, will become less frequent. The first point mentioned, however, namely, the performance of a suitable operation, needs to be expanded. After our experience in Neyyoor we have been forced more and more to the conclusion that for all cases for which a gastro-enterostomy is contra-indicated by pyloric permeability or hyperacidity, a gastrectomy of the Finsterer type is the ideal operation.

GASTRECTOMY

The functions of a gastrectomy are ideally:—

1. To lower gastric acidity. All gastric operations do this for a short time, and Polya or Billroth II may do so for some months. But the ideal gastrectomy from this point of view will remove a large proportion of the acid-secreting portion of the stomach, and thus effect an actual substantial diminution of gastric acidity.

2. To create a one-way traffic system, denying the food all possible access to the ulcer.

3. To promote healing of the ulcer either by the actual removal of it, or by leaving it in a condition in which it will heal.

The leaving behind of a portion of the pyloric end of the stomach (*Fig. 181, A, 4*) will result in a free secretion of mucus in this region, with which the ulcer is surrounded. In practice, we have found less trouble and pain after this operation than after a simple gastro-enterostomy. Incidentally, this mucus will be of benefit to the intestine at the region of the anastomosis, in decreasing the likelihood of recurrent ulcer. Also, it is easier to divide the stomach a little distance away from the pylorus rather than close to it. Several of our deaths from gastrectomy were due to the great difficulty in closing a pylorus near the sphincter; a couple of inches away from it there is no difficulty whatever.

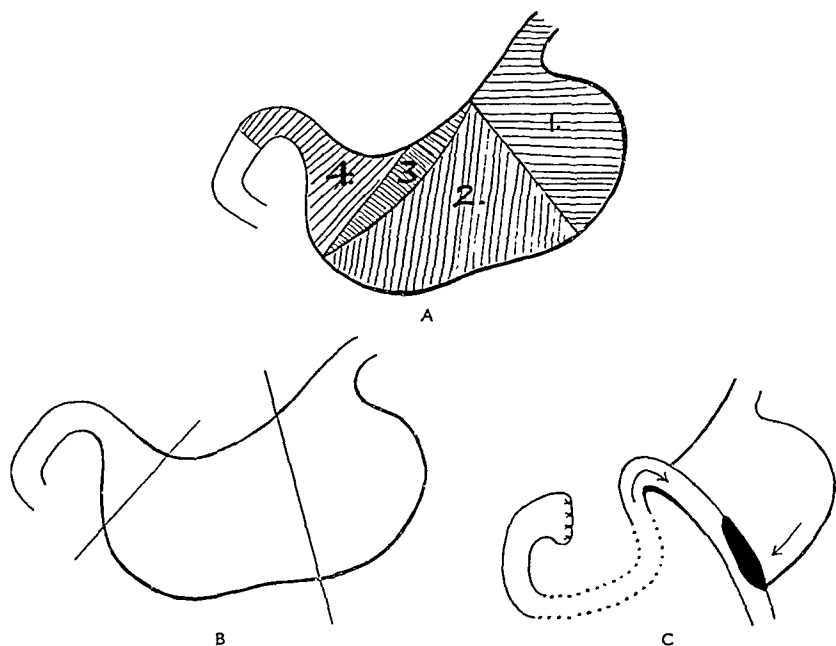


FIG. 181.—A, Shows the physiological divisions of the stomach: 1, Cardiac portion; 2, Fundus; 3, 'Gastric pathway'; 4, Pyloric antrum. B, Shows the amount of stomach removed in the Finsterer gastrectomy. C, The re-formation of the stomach after gastrectomy.

We have entirely given up all thought of removing duodenal or pyloric ulcers, for the same reason. They invariably heal if one-way traffic is established by a gastrectomy; and every surgeon knows that excision of a duodenal ulcer can be one of the most precarious and difficult of all abdominal operations.

Some surgeons still consider that so severe an operation as gastrectomy should not be performed for duodenal ulcer. The answer is two-fold: first, that in proper hands gastrectomy is not an operation with high mortality—as a primary operation for duodenal ulcer we have done it 81 times with but 2 deaths; second, that under certain conditions gastro-enterostomy carries a higher risk.

It will be seen from *Table II* that the greater part of the 6.5 per cent mortality of gastrectomy is when that operation is performed for gastric or gastro-jejunal ulceration. A gastrectomy performed on a stomach which is not itself ulcerated, adherent, or inflamed, is not, if conducted properly, a dangerous operation. Surely an operation with a 2 or 3 per cent risk is preferable to the alternative anastomosis

with an operative risk of 1·5 per cent and an ulcer-recurrence rate of 6 per cent, to say nothing of the other 10 per cent of cases who have trouble of some sort after gastro-enterostomy.

Table II.—GASTRECTOMIES DONE AT NEYYOOR, 1925-35

DISEASE		NUMBER OF OPERATIONS	DEATHS	PERCENTAGE
Duodenal ulcer	81	2	1·3
Gastric ulcer	46	5	11·0
Gastric carcinoma	18	1	5·5
Gastro-jejunal ulcer	57	5	9·0
Total	202	13	6·5

The operation now performed at Neyyoor for reduction of acidity is therefore the high gastrectomy associated with Finsterer, and its effects are :—

1. Permanent reduction of acidity by the removal of a large area of the fundus.*
2. Exclusion of the ulcer from irritation by food and promotion of its healing by the mucus secreted in the pyloric antrum.
3. No risk of leakage from an imperfectly closed duodenum.
4. No risk of regurgitation of food into the duodenal blind end, owing to the vertical position of the stoma.

It must be stressed at this point that this high gastrectomy operation is not to be a routine treatment for all duodenal ulcers. Where there is stenosis, or where there is low or normal acidity, gastro-enterostomy remains the operation of choice.

Where, however, there is very high acidity and rapid emptying of the stomach, the high Finsterer gastrectomy is indicated. One of us (I. M. O.) has recently done 30 cases of these operations without a death, in cases which included 9 gastro-jejunal ulcers and several poor operative risks. In this series there were no cases of bilious vomiting, and the convalescence has been remarkably smooth—better, in fact, than after the average gastro-enterostomy. It is too early to report late results from this type of operation, as it is only recently that we have been driven by all the foregoing conclusions to perform it. All that we have heard about are in a satisfactory condition, except for one case of anæmia, in a woman who had a gastric ulcer, with anæmia and low acidity at the time of operation. If the acidity is high at the time of operation, and reduced by operation to normal or thereabouts, the danger of anæmia is almost nil.

THE TREATMENT OF GASTRO-JEJUNAL ULCER

Here we are up against an ulcer caused presumably by the same causes as produced the original duodenal ulcer—acidity, low vitamin A diet, irritating food-stuffs, sepsis—with the possible addition of faulty technique in the original operation.

The particulars of 72 cases treated at Neyyoor are set out in Table III.

* Note.—We have proved by test-meal records that the reduction in acidity varies with the amount of stomach removed. In the few cases in which one of us (I. M. O.) has carried out test-meals several months after the operation, it is found that the reduction in acidity is maintained.

Table III.—GASTRO-JEJUNAL ULCERS TREATED AT NEYYOOR, 1925-35

Note.—PGE = Posterior gastro-enterostomy; AGE = Anterior gastro-enterostomy; GJ = Gastro-jejunal ulcer; G = Gastric ulcer; D = Duodenal ulcer; J = Jejunal ulcer; C = Gastrocolic fistula; B = Billroth; P = Polya; F = Finsterer high gastrectomy; Y = Anastomosis (jejuno-jejunal); E = Excision of ulcer; A = Anterior.

No. OR DATE	ORIGINAL OPERATION	INTERVAL	COMPLICA- TION	SECOND OPERATION	REMARKS
446	PGE	2½ yr.	GJ	E	Suspected to have a recurrence of the ulcer when seen 8 years later
320	PGE	3 yr.	GJ	E	Satisfactory
308	PGE	2 yr.	GJ	P	Pain later due to kink, relieved by entero-anastomosis
557	PGE	9 m.	GJ	E	Satisfactory
520	PGE	3 yr.	GJ	B II	Satisfactory
522	B I	1 yr.	GJ	B II	Secondary ulcer in jejunum, excised 1 month later. Satisfactory result and rapid gain in weight
768	PGE	?	GJ	E + Y	Satisfactory—not followed-up
781	PGE	3½ yr.	GJ	E + Y	Continuance of pain
882	PGE	1 yr.	GJ	E	Died
888	PGE	5 m.	GJ	E	Satisfactory
717	PGE	4 yr.	GJ	E	Unsatisfactory: continued pain
893	PGE	1 yr.	GJ	E	Immediate result satisfactory
1929-6	PGE	3 yr.	GJ	E	Immediate result satisfactory
1929-9	PGE	1½ yr.	GJ	E	Immediate result satisfactory
1927-II	PGE	2 yr.	GJ	E + AGE in Y	Immediate result satisfactory
1929-3	PGE	4 yr.	G + D	P	Immediate result satisfactory
1325	PGE	2 yr.	GJ + C	AGE + Y	Satisfactory 1 year later
1350	PGE	1 yr.	GJ	B II	Improved
1932-10	PGE	2 yr.	GJ	P + Y	Satisfactory
1932-10	PGE	1 m.	GJ	P + Y	Satisfactory
1028	PGE	2 yr.	GJ	E	Satisfactory
1036	PGE	1 m.	GJ	P + Y	Satisfactory
1126	PGE	1 m.	GJ	E	Unsatisfactory: recurrence of ulcer suspected
1130	PGE	6 m.	GJ + G	E	Satisfactory
1174	PGE	4 m.	GJ	E	Recurrence of GJ ulcer, relieved 2½ yr. later by B II
1190	PGE	4 yr.	GJ	E	Recurrence of GJ ulcer, relieved 1½ yr. later by B II
1209	PGE	2 yr.	GJ	E	Satisfactory
1261	PGE	3 yr.	GJ	B II	Satisfactory
1344	PGE	3 yr.	GJ + C	E	Stormy convalescence, but now satisfactory
1359	PGE	1½ yr.	GJ	P + Y	Satisfactory
1388	PGE	2 yr.	GJ	E + Y	Recurrence of GJ ulcer, apparently cured medically
1454	PGE	2 yr.	2 GJ's	P	Died—subphrenic abscess
1457	PGE	6 m.	GJ + C	P	Satisfactory
1518	PGE	4 yr.	GJ	B II	
1589	PGE	4 yr.	GJ + C	B II	Satisfactory
1590	PGE	6 yr.	GJ + C	E	Died
1620	PGE	6 m.	3 G's	B II	Satisfactory
1646	PGE	3 yr.	G	B II	Satisfactory
1654	PGE	8 yr.	J	E etc.	Satisfactory after PGE and AGE + anastomosis
1688	PGE	4 yr.	G	P	Died
1692	PGE	1 yr.	GJ + C	P + Y	Satisfactory
1736	PGE	1½ yr.	GJ	B II	Satisfactory—silk thread in PGE suture found
1758	PGE	1½ yr.	GJ	B II	Satisfactory

(Continued on next page)

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Table III.—GASTRO-JEJUNAL ULCERS TREATED AT NEYYOOR, 1925-35, continued

No OR DATE	ORIGINAL OPERATION	INTERVAL	COMPLICATION	SECOND OPERATION	REMARKS
1762	PGE	2½ yr.	GJ	P - Y	Satisfactory: PGE found done the wrong way round
1773	PGE	5 yr.	GJ	P - Y	Died
1809	PGE	6 m.	GJ	B II	Satisfactory: silk thread as No. 1736
1891	PGE	4 yr.	GJ	B II	Re-operated 5 m. later for intussusception of small intestine. Recovery
1901	PGE	6 yr.	GJ + J	P - Y	Satisfactory
1903	PGE	7 m.	GJ	E	Bad position of original operation corrected. Hence no gastrectomy
1926	PGE	3 yr.	J + C	E - B II	Satisfactory
1930	PGE	6 yr.	G	B II	Satisfactory
1933	PGE	3 yr.	J	P - Y	Satisfactory
1965	PGE	7 yr.	J + C	E + B II	Satisfactory
1969	PGE	8 yr.	GJ	B II	Satisfactory
2005	PGE	3 yr.	GJ	E	Satisfactory, but pain and tenderness over stoma; X rays show no abnormality
2028	PGE	4 yr.	GJ	P - Y	Satisfactory
2073	PGE	1 m.	GJ	B II	Satisfactory: slight discomfort daily
2082	PGE	9 yr.	GJ	P	Satisfactory
2152	PGE	6 yr.	GJ	B II	Satisfactory
2226	PGE	1 m.	GJ	E	Satisfactory
2230	PGE	5 m.	GJ	B II	Satisfactory except for slight pain over stoma 1 yr. later
2233	PGE	1 yr.	G	P	Satisfactory
2285	PGE	1 yr.	GJ	E + B II	Satisfactory
2293	PGE	7 yr.	GJ	E	Satisfactory
2300	PGE	11 days	GJ	P - Y	Died from leakage
2313	PGE	4 yr.	GJ	P - Y	Died
2321	PGE	?	GJ	E - B II	Satisfactory
2344	PGE	4 yr.	GJ + J	P	Satisfactory
2334	PGE	8 yr.	2 D's	B II	Good at first, but died 2 m. later
2372	PGE	6 m.	GJ	F	Satisfactory
2382	PGE	?	GJ	P	Satisfactory
2383	PGE	?	GJ + C	P - Y	Died of cerebral œdema
2476	PGE	12 yr.	GJ	P - Y	Satisfactory

Analysis.—Out of 2500 operations for gastric, duodenal, and jejunal ulcers:—

OPERATIONS FOR GASTRO-JEJUNAL ULCERS	No OF CASES	DEATHS	RECURRENCE	UNSATISFACTORY
Excision of ulcer and re-forming stoma	24	4 (16.6%)	8 (33.3%)	—
Billroth II	28	—	1	2
Polya	20	5 (25%)	1 (5%)	1
Totals	72	9 (12.5%)	10 (13.8%)	3 (4%)

Since these statistics were compiled, one of us (I. M. O.) has done high (Finsterer type) gastrectomy in 9 cases of gastro-jejunal ulcer with no deaths, making a total of 81 cases, 9 deaths (11.1 per cent). Two of these cases, as mentioned elsewhere, were recurrences of ulcer after a low gastrectomy (B II). Of the 72 cases tabulated, 35 had had the primary operation performed at Neyyoor, the remainder were from other surgeons.

From this table it will be seen :—

1. That the operation of excision of the ulcer and re-formation of a gastro-enterostomy has been given up as producing unsatisfactory results. It is difficult to do, and leaves the mechanics of the digestive apparatus in the same condition as they were when the recurrent ulcer was produced, and is manifestly unsound.

2. That the Billroth II operation is less risky than one of the Polya type. This is doubtless partly due to the fact that the smaller the ulcer, the more likely is it possible to do the Billroth II operation, whereas if there is much induration, etc., a Billroth II will be impossible.

3. That the average mortality of these cases was over 10 per cent.

4. That since the high gastrectomy operation has been done, this mortality has been reduced.

It is sufficient to say that it is obvious that the best thing that can be done for a gastro-jejunal ulcer is that which is most likely to prevent its recurrence in the first place. As in the case of duodenal ulcer, we are driven to the conclusion that an excision of the ulcer followed by a high Finsterer gastrectomy is the ideal treatment. For the cases which get gastro-jejunal ulcers are just those with high acidity, etc.,

which ought many of them to have been treated originally by means of a high gastrectomy. In dealing with many of these cases one is up against a major surgical problem, and one that may tax the skill of any operator.

Adherence to the colon may complicate matters, but so long as there is no fistula it need not be serious. The base of a gastro-jejunal ulcer may be left on the outside of a colon, and as the anastomosis, together with the fundus of the stomach, is going

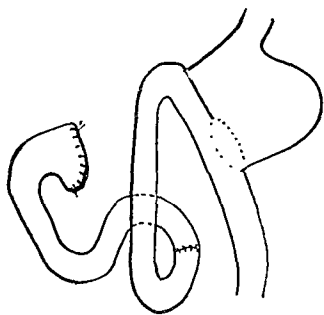


FIG. 182.—Method of re-formation of intestine, and performance of gastrectomy operation by method which does not involve a Y-anastomosis.

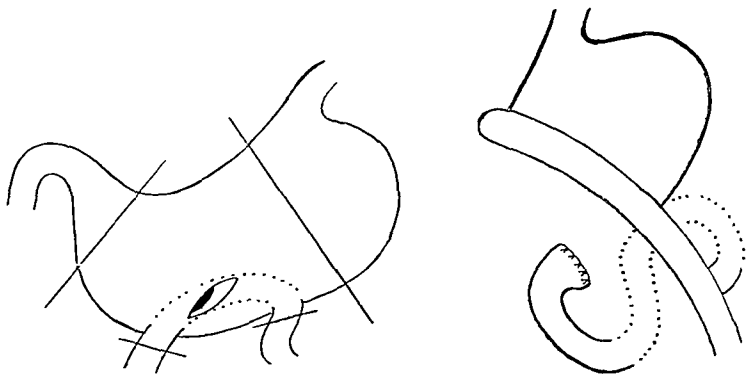


FIG. 183.—The amount of stomach removed in the gastrectomy for gastro-jejunal ulcer and the method of re-formation of the stomach.

to be excised in any case, mutilation of the anastomosis or its area does not matter. The ulcer-base on the colon is then covered by omentum and should cause no trouble.

Having decided that a high gastrectomy is necessary, there are two ways in which it may be done :—

1. The jejunum may be joined end to end, and a Finsterer gastrectomy performed (*Fig. 182*). This is the method of choice, as it gives a chance for the bile and mucus from the duodenum to get into the stomach and help to reduce its acidity and to protect the mucosa of the jejunum near the anastomosis.

2. The gastrectomy may be performed in Y, as in *Fig. 183*. We have done this on several occasions, but theoretically it is not so sound as the other method.

The essential feature of the operation in either case is the removal of a large part of the acid-secreting portion of the stomach, and the preservation of the pyloric antrum; and although it is radical, it has not yet shown itself to have a high mortality, and compares favourably in this respect with all the other methods of treatment. We hope to report further in a few years' time on the late results of this type of operation compared with those of other methods.

CONCLUSIONS

1. In South India, lack of vitamin A and other dietetic factors seem to be the principal cause of duodenal ulceration, and it is possible that these factors have been insufficiently considered as causative of duodenal ulcer in the Western countries.

2. Gastro-enterostomy is the operation of choice for duodenal ulcer where there is stenosis.

3. High gastrectomy, without excision of the pyloric antrum, is the operation of choice for gastric and gastro-jejunal ulcer, and for duodenal ulcer with high acidity and rapid emptying.

REFERENCES

- ¹ KONJETZNY, *Arch. f. klin. Chir.*, 1924, cxxix, 139.
- ² PUHL, *Med. Klin.*, 1927, xxiii, 986, 1063.
- ³ CARSWELL, *Atlas of Pathol. Anat.*, 1838, reproduced in Hurst & Stewart, *Gastric and Duodenal Ulcer*, 1929. Oxford Med. Pub.
- ⁴ FABER, *Lancet*, 1927, ii, 901.
- ⁵ HURST, *Guy's Hosp. Rep.*, 1921, lxxi, 450.
- ⁶ BARCLAY, *The Alimentary Tract*, 1915. London.
- ⁷ BOLTON, *Ulcer of the Stomach*, 1913. London.
- ⁸ HURST AND STEWART, *Gastric and Duodenal Ulcer*, 1929, 59, 60. Oxford.
- ⁹ ROSENOW, *K. Inf. dis.*, 1923, xxxiii, 248.
- ¹⁰ MAINGOT, *Post-Graduate Surgery*, i, 184.
- ¹¹ MCCARRISON, *Ind. Jour. Med. Research*, 1931, and *Brit. Med. Jour.*, 1931, i, 970.
- ¹² See also HARRIS, *Jour. Amer. Med. Assoc.*
- ¹³ MAINGOT, *Op. cit.*, 404.
- ¹⁴ GARNETT WRIGHT, *Brit. Jour. Surg.*, 1935, xxii, 435.
- ¹⁵ MORLEY, *Ibid.*, 1928, xvi, 247.
- ¹⁶ GORDON TAYLOR, *Ibid.*, 1929, xvi, 659.
- ¹⁷ LAKE, *Lancet*, 1928, ii, 268.

CONGENITAL DISC-SHAPED LATERAL MENISCUS WITH SNAPPING KNEE*

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THE occasional persistence of the embryonic disc shape of the lateral meniscus of the knee-joint has been known to anatomists for many years. The first dissecting-room specimen to be described was that by Young of Glasgow in 1889, and since then twelve examples have been placed on record.

Several authors have described the development of the cartilage fully and pointed out the relationship between its embryonic disc shape and the disc-shaped meniscus seen in the adult. Attention has also been directed to the appearance of a disc- or ring-shaped meniscus in certain lower animals such as monkeys and some lizards, though it is doubtful whether these comparative studies have much bearing on the condition in the human being. It would be a work of supererogation to detail this work, as it has been so ably summarized by Jaroschy of Prague, whose work on congenital disc cartilage will always remain the classical study of this interesting subject.

The occurrence of a striking snapping noise in the knee-joint on movement, along with a visible separation of the femur and tibia, has been noted on many occasions in European literature. European journals before 1914 are strewn with examples of such cases. The etiology was found puzzling owing to the absence of previous trauma, and the occasional occurrence of a similar snap in the knee-joints of children very soon after birth. These cases were not submitted to operation, and the snapping was as a rule attributed to a 'subluxation' owing to laxity of the capsule and ligaments of the joint. From the clear-cut clinical histories accompanying these cases, however, there can be no doubt that they were examples of snapping knee, due to congenital disc-shaped meniscus.

It was in 1910 that the association of a disc-shaped lateral meniscus with snapping knee was first pointed out by Kroiss of Innsbruck, who removed from a snapping knee a torn lateral meniscus and appreciated the importance of the fact that the meniscus, though torn, was so much wider than normal as to come into the class of what is now recognized as a disc-shaped meniscus. Since 1928, starting with one case by Bristow and two by Schulz, a number of examples have been described, and longer articles have appeared by Finder, Bell-Jones, Jaroschy, and Timbrell Fisher. I have already alluded to the importance of the very complete historical, clinical, and anatomical study of Jaroschy. Up to the present date 30 fully documented examples of snapping knee from this cause have appeared in the literature, along with 19 more which are mentioned as having been encountered without any clinical or anatomical detail being given. The fact, however, is that

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49 cases of disc-shaped meniscus causing snapping knee have been treated by operation since 1910, and 48 of these have been published since 1928.

Timbrell Fisher in a recent article draws a parallel between this condition and cysts of the meniscus. He points out that these cysts were at one time looked upon as rarities, but that as knowledge of the condition became diffused they became progressively more common and are now met with so frequently as not to call for comment. Fisher believes with justice that the same course will be followed by disc menisci, and that their apparent comparative rarity is mainly attributable to the fact that they are not sufficiently often recognized.

It cannot be gainsaid that, though all orthopædists and those interested in the surgery of the knee-joint are conversant with the condition, and most of them have operated on a varying number of cases, this type of snapping knee, as an entity, is still largely unknown to the general surgeon.

During the past two years I have met with 4 examples of snapping knee of this type, and have also had the opportunity of examining one dissecting-room specimen. A survey of these cases has led me to certain conclusions regarding the mode of production of the snap, a subject which has been largely ignored by previous authors.

A chronological list of references to previously recorded cases is appended at the end of this paper.

CASE REPORTS

Case 1.—G. G., male, aged 11 years.

HISTORY.—Six weeks before admission to The Church of Scotland Deaconess Hospital, the patient was swinging his legs while sitting in a chair, when he felt a sudden acute pain over the outer side of the right knee. Before this, he had had no trouble with the joint. Afterwards the movements of the knee were painful for a few days, but no locking occurred. He rested in bed for a few days and when he started to go about again he noticed a definite 'clicking' noise in the knee. This clicking was at times associated with a little swelling of the joint and vague pain and instability.

ON EXAMINATION.—There was a trace of effusion into the right knee. No wasting of the quadriceps was present. On movement of the joint, a marked click or snapping sensation could be elicited at points just short of full flexion and of full extension. There was no history of knee trouble in parents or relatives, and X-ray examination was negative.

OPERATION, Nov. 11, 1933.—The joint was opened in front of and behind the fibular collateral ligament by a lateral Timbrell Fisher incision convex distally. The lateral meniscus was seen to be very broad, filling the whole lateral compartment of the joint, and it was split longitudinally. The meniscus was removed in several pieces and the specimen was unfortunately destroyed.

Convalescence was uneventful, though delayed by a slight post-operative effusion. Re-examination of the patient in November, 1935, showed the knee to be perfectly stable, and function appeared to be completely normal.

Case 2.—A. N., male, aged 13 years. (*Fig. 184.*)

HISTORY.—According to the patient and to his parents, the right knee had been the seat of a "cracking" sound "all his life." At times he had suffered from slight discomfort and a little effusion into the joint, but it had never seriously interfered with his activities. There was no history of injury to the joint.

ON EXAMINATION.—There was no visible abnormality in the joint and no wasting of the quadriceps was present. On flexing the joint, at a point some 20° to 30° short of full flexion there was a slight 'check' in the smooth movement of the joint; the femoral and tibial condyles appeared to separate from each other momentarily, and as they fell together again an audible and palpable 'clunking' noise was evident. Extension from the fully flexed

position was perfectly smooth up to a point some 20° short of full extension, when the 'clunking' noise repeated itself. The snapping was completely painless. There was no history of any knee troubles in the family, and the left knee appeared normal on clinical examination. X-ray examination showed no obvious abnormality.

OPERATION, Jan. 25, 1934.—At operation in The Church of Scotland Deaconess Hospital the knee-joint was opened by a wide lateral Timbrell Fisher incision, reflecting the patella medially in order to get a free view of the offending cartilage during movement of the joint. A drawing was made of the joint at this stage (Fig. 184). A wide cartilage was found covering the whole lateral condyle of the tibia, save for a small area nearly half an inch in diameter close to the intercondylar ridge. In front of this bare area, the anterior horn of the broad cartilage swelled out to blend widely with the intercondylar structures, while the posterior

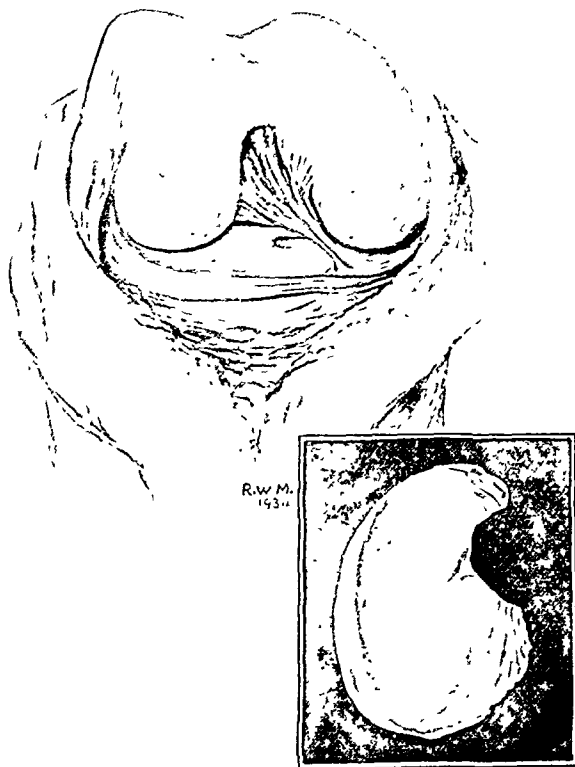


FIG. 184.—Case 2. A drawing of the knee-joint made at the time of operation. The broad disc-shaped lateral meniscus can be seen, and the inset shows the meniscus after removal, with a definite oblique ridge upon the upper surface.

horn, though somewhat wide, was much more normal in shape. The upper surface of the meniscus presented a well-marked ridge running obliquely laterally and forward from the notch on the medial border. This ridge divided the femoral surface into two portions or facets—an anterior one which articulated with the femoral condyle in extension and a posterior one articulating with the condyle in full flexion.

On flexing and extending the joint the snap could be reproduced. Starting with the joint in full flexion, and the meniscus largely visible in front of the condyle of the femur, which was in contact with the posterior facet on the meniscus, on extension the meniscus was seen to become gradually more and more bunched up in front of the femoral condyle as the articular surface of the latter rolled forwards on to the ridge. At a point 20° short of full extension, the femoral condyle could be seen to ride up on the ridge, causing the

visible separation of the femoral and tibial condyles which has been stressed so frequently by previous authors. The femoral condyle then slipped suddenly over the crest of the ridge, and sank down with an audible 'clunk' or 'thud' into the anterior facet on the meniscus in front of the ridge. On flexion the process was reversed, the femur riding over the ridge into the posterior facet short of full flexion.

The meniscus was removed entire and the joint closed.

Convalescence was uninterrupted, and on examination in December, 1935, all signs of snapping had disappeared and the function of the joint appeared to be normal in all respects.

Case 3.—I. T., male, aged 13 years. (Fig. 185.)

HISTORY.—At the age of four this patient was confined to bed for a period of two years with a tuberculous spine, making a good recovery. On getting up and resuming walking, a snapping noise was noticed in his left knee on movement. The noise was always present and never varied. There was no history of knee trouble in other members of the family.

ON EXAMINATION.—A 'clunking' noise and sensation, similar in all respects to that described in the previous case, could be elicited on movement of the joint at points just short of full flexion and of full extension of the joint. There was no swelling of the joint and no quadriceps wasting.

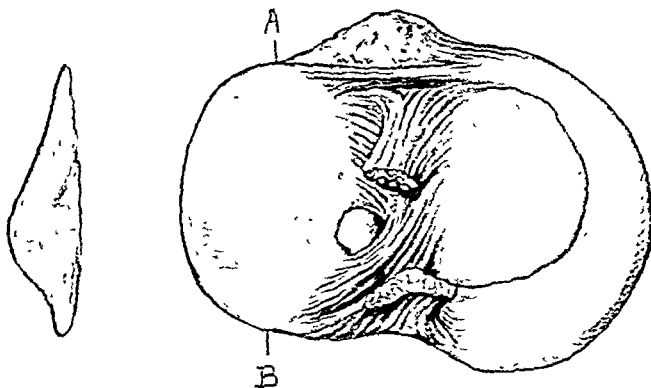


FIG. 185.—Case 3. The drawing shows the position occupied at operation by the lateral meniscus, with its broad disc shape and the well-marked oblique ridge over which the femur rode to produce the snapping. The height of the ridge is well brought out in the inset, which shows the longitudinal section of the meniscus from A to B.

OPERATION, April 18, 1935.—At operation in The Church of Scotland Deaconess Hospital the left knee-joint was opened by a lateral Timbrell Fisher incision, reflecting the patella inwards. The lateral meniscus was observed to have a roughly quadrilateral shape with a small notch on its medial margin. It formed a complete disc covering the lateral condyle of the tibia entirely save for a small area about a quarter of an inch in diameter close to the intercondylar area. Both anterior and posterior horns of the meniscus were broad and thick and blended widely with the structures attached to the intercondylar area. The femoral surface of the meniscus was marked by a well-formed oblique ridge running laterally and forwards from the region of the small intercondylar notch. This ridge divided the meniscus into anterior and posterior facets, and on movement of the joint it was observed that the femoral condyle rode over the ridge at points just short of full extension and full flexion, occupying the anterior facet in full extension and the posterior one on full flexion. The meniscus was removed entire, and the inset in Fig. 185 shows the well-marked ridge on longitudinal section of the meniscus.

Convalescence was uneventful, and examination of the patient in December, 1935, showed that the snap had completely disappeared and the knee-joint was normal in every way.

Case 4.—F. D., female, aged 18 years. (Fig. 186.)

HISTORY.—Two years previously the patient, who had been in employment in a rubber works, had noticed the gradual commencement of a snapping noise in the left knee. She

is sure that there was never any injury and that she had never had any pain in the knee whatsoever. Apart from the snapping, which annoyed her slightly, she had no trouble with her knee, and actually came to hospital on account of a sprained wrist, merely mentioning the condition of her knee incidentally.

ON EXAMINATION.—The knee appeared to be perfectly normal, and there was no wasting of the quadriceps or local tenderness. On movement of the joint a typical 'clunking' noise

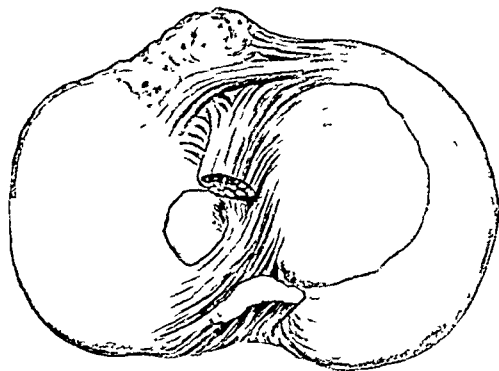


FIG. 186—Case 4. A drawing to show the relationship of the lateral meniscus as seen at operation. The meniscus is disc-shaped, and separated round the periphery of its anterior horn.

was elicited a few degrees short of full flexion and extension, the syndrome being indistinguishable from that described in the previous cases. X-ray examination was negative.

OPERATION, Aug. 16, 1934.—At operation in The Church of Scotland Deaconess Hospital the left knee was opened by a lateral Timbrell Fisher incision, reflecting the patella medially. The lateral meniscus was seen to be of the typical disc shape, with a broad anterior horn

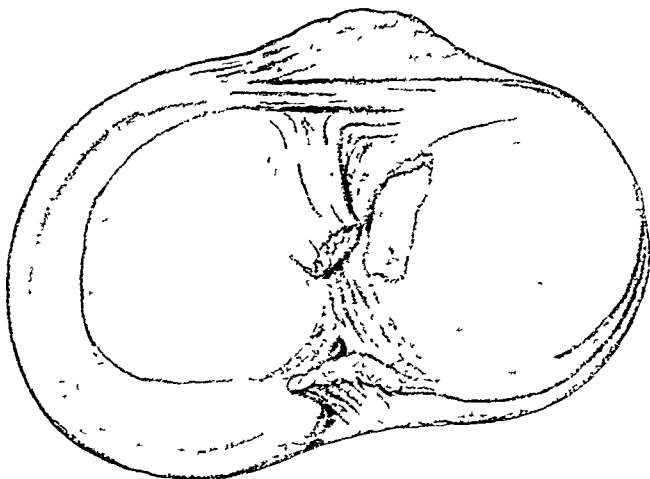


FIG. 187—Anatomical specimen, by courtesy of Dr. G. J. Boyd. No history available, but the abnormality was discovered in the dissecting room. The lateral meniscus is disc-shaped, and presents a smooth concave upper surface. There is no reason to suppose that such a meniscus had caused snapping during life.

blending with the intercondylar structures. Between the two cornua, there was a small notch about a quarter of an inch across through which the upper aspect of the lateral tibial condyle could be seen. The periphery of the anterior horn of the meniscus was separated from its attachments. There was no ridge present on the upper aspect of the meniscus, this surface being a smooth concavity.

On movement, the femoral condyle was observed to occupy the concavity of the meniscus in the position of flexion, while as the position of extension was approached the separated anterior horn slipped backwards behind the condyle, which then lay in direct contact with the upper aspect of the tibia. The reverse occurred at a point near full flexion, the meniscus suddenly jumping forwards again between the femur and tibia. The 'clunking' noise was caused by the sudden movement of the femoral condyle backwards or forwards over the thick free anterior edge of the meniscus.

Anatomical Specimen.—By courtesy of Dr. G. J. Boyd. (*Fig. 187.*)

This specimen was encountered in the dissecting rooms of the University of Leeds. No history is forthcoming. The specimen consisted of the upper portion of the tibia and fibula of an adult with the menisci in situ. The medial meniscus is normal. The lateral meniscus forms an almost complete disc, covering the upper aspect of the lateral tibial condyle completely save for a small elongated area close to the intercondylar area. The upper aspect of the meniscus is smooth and concave, there being no ridge present. The specimen is somewhat hard, as befits one emanating from the dissecting room, but there is nothing to suggest that the meniscus was unduly mobile during life. A slight degree of separation of the anterior horn has evidently been the result of dissection. *Fig. 187* shows the cartilage, the whole appearance of which suggests that it represents a disc-shaped meniscus which has remained symptomless throughout life.

DISCUSSION

A snapping noise in the outer part of the knee-joint has long been associated with lesions of the lateral meniscus, and forms indeed a common picture in tears of that structure. The classical case of non-traumatic snapping knee due to congenital disc-shaped lateral meniscus may simulate a torn lateral meniscus very closely, and it should be realized at once that almost half of the cases of disc meniscus on record are examples of tearing of a malformed disc, and also that abnormal wideness or complete disc formation is present in a large proportion of the ordinary traumatic lesions of the lateral meniscus (18 per cent according to Bell-Jones). In other words, the disc meniscus is more liable to injury than the normal one.

In cases of non-traumatic snapping knee from disc meniscus, snapping may be noted at or soon after birth, but frequently appears later in life without any obvious pre-existing injury. The cases in extreme infancy are unusual, but I am conscious of having in the past seen two cases of snapping knee within the first two or three months of life where the symptom had been noted by the parents soon after birth. It is important to note that when snapping occurs at this early age, and indeed even in later life, it may pass off entirely after having been present for a considerable time.

In such a developmental abnormality as this it is almost certain that inheritance plays a part in its transmission. In one case recorded by Bell-Jones, indeed, the mother of the affected child showed similar symptoms, and though the state of the parental knee-joint was not investigated by operation there can be little doubt that she presented a disc meniscus. There was no family history in my personal cases, but having regard to the fact that a large proportion of disc menisci are probably symptomless throughout life and that the total of recorded cases where a history of any sort is available is small, it is not surprising that only one case with a history of familial transmission is on record. It would be interesting to accept the case described by von Stackelberg in 1927 of snapping knee, with associated deformities, including partial absence of the tibia, as due to a disc

cartilage, but Jaroschy writing in 1935 hesitates to do so. Certainly the illustrations in a later article by von Stackelberg (1929), where dissections of the knee are shown, do not warrant its inclusion as an example of the type of snapping knee under consideration.

Clinical Features.—These, when present, consist of a painless snapping sensation on movement of the affected joint. Where only one knee is the seat of snapping, the disc formation may be bilateral. Snapping does not seem to be the cause of much disability in non-traumatic cases, though complaints of occasional vague pain and slight synovial effusion may be met with.

On examination, the configuration of the joint is normal, and there is no muscular wasting in the quadriceps extensor apparatus in the absence of super-added injury to the meniscus.

The typical 'snap' takes place at the extremes of movement, occurring usually at points about 20° short of full flexion and of full extension of the joint. When the snap occurs, a momentary check in the smooth movement of the joint is seen, and the tibia and femur appear to separate slightly on the outer side of the joint and then come together again. At the same time it is said that a slight protuberance appears between them which is thought to be the meniscus itself. This check in movement and separation of the lateral condyles is momentary, and is accompanied by a palpable and audible noise. The noise has been described as a 'snap', 'click', 'thud', or 'clunk', the last being, perhaps, the most descriptive. Finner believes that radiographic examination shows an aplasia of the lateral condyle of the tibia and a widening of the lateral joint space, but I have been unable to convince myself of the diagnostic value of this appearance in my own cases.

The above description of the clinical features of snapping knee appears to be fairly constant for non-traumatic cases. Where, however, a previously symptomless disc meniscus suffers from a superadded tear, symptoms may be caused which are indistinguishable from the non-traumatic case, as in my first case, or the less obvious snapping of a torn lateral meniscus may appear. Not infrequently snapping may be completely absent in such traumatic cases, and a diagnosis may rest upon the history of injury along with quadriceps wasting and finger-point tenderness over the torn meniscus.

Anatomy of the Disc Meniscus.—This varies comparatively little from case to case. In some examples the general form is circular and the upper surface is concave, as in the dissecting-room specimen which I have described (*see Fig. 187*). More commonly the disc is almost quadrilateral in shape with a small notch on its medial margin. Here one gets the impression that not only is the body of the meniscus broad, but that both cornua are greatly broadened and have wide attachments to the intercondylar structures, as in *Fig. 185*. Finally, there is what may be called the 'comma-shaped' meniscus seen in *Fig. 184*, where the body and the anterior cornu of the meniscus are broadened while the posterior horn remains almost within normal limits. It is quite likely that a similar condition affecting the posterior horn only may occur, though I have not seen a case figured in the literature. The circular, quadrilateral, and anterior-horn 'comma-shaped' menisci are, however, all represented in the illustrations of other authors.

Etiology of the Snapping Noise.—The mere presence of a disc meniscus does not cause symptoms, but symptoms requiring removal of the structure may be brought about in several ways.

1. *Trauma*.—The disc-shaped meniscus is undoubtedly more liable to injury than the normal lateral meniscus, as explained above.

2. *The Ridged Disc*.—In this class, to which *Cases 2 and 3* belong, a ridge develops on the upper aspect of the meniscus with a concave facet in front of and behind it. In flexion of the joint the condyle of the femur lies behind the ridge, and on extension taking place between the femur and the meniscus the latter structure becomes bunched up in front of the advancing condyle until late in the movement. At a point about 20° short of full extension, however, the femur mounts the ridge, rides over the crest, and comes to occupy the anterior facet, producing the classical signs as it does so. A similar but reversed process takes place during flexion of the joint, the snap occurring about 20° short of full flexion (Fig. 188). A similar backward and forward movement to this has been noted in cases described by Schulz and Jaroschy, though the importance of the ridge does

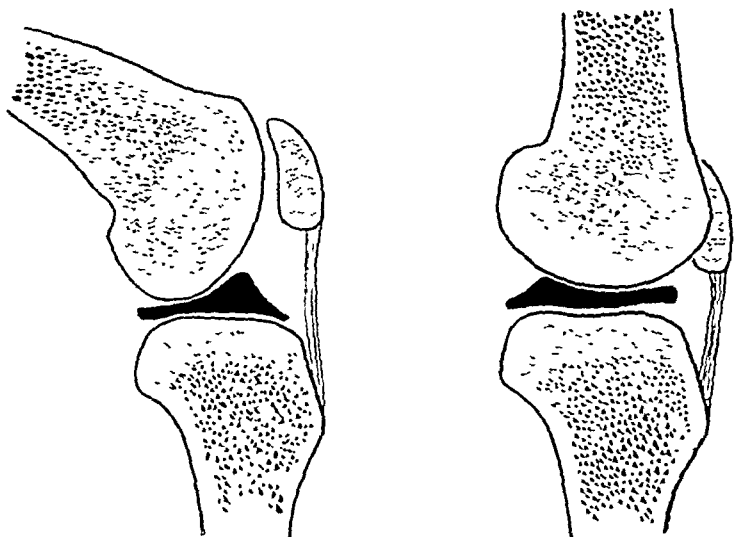


FIG 188.—Diagram to show the way in which a ridged meniscus causes snapping, and the relationship of the femoral condyle to the meniscus and ridge in flexion and in extension.

not appear to have been appreciated. In another case of Jaroschy's the meniscus is described as passing backwards in flexion and forwards in extension, a reversal of the usual movement.

3. *Abnormal Mobility of the Disc*.—The normal lateral meniscus moves backwards and forwards fairly freely on flexion and extension of the knee-joint, but eventually the range of movement of the disc meniscus may become so great and the anterior or posterior attachments of the meniscus so stretched that the whole meniscus may slip backwards and forwards in flexion and extension, causing snapping. In *Case 4* the anterior attachments had become separated entirely in the absence of any trauma. Here in flexion the femoral condyle articulated with the upper aspect of the disc in the normal fashion, while during extension the condyle rolled and slipped over the free anterior margin with a thudding sound, the disc meniscus disappearing behind the condyle, which then came to articulate directly with the tibia.

There are cases on record where the posterior horn has become separated, and it is probable that in these cases the process was reversed.

Treatment.—In most cases of persistent snapping due to disc menisci the offending meniscus should be removed. In young children operation may be delayed, as the snapping not infrequently disappears spontaneously. In older children this is less likely to happen, and there can be no doubt that prolonged snapping with the slight effusion and synovial reddening which accompany it may eventually, if untreated, lead to the development of osteo-arthritis.

The meniscus is easily removed through the ordinary short incision in common use for traumatic lesions of the menisci in this country. The long patella-reflecting incision was utilized in my cases for the sole purpose of being able to watch the cartilage easily on movement and to work out the mechanism of the snapping sound.

It will be remembered that on full flexion the snapping disc jumps forwards into the anterior portion of the joint, and remains there until a point not far short of full extension, when it jumps backwards. During the application of a tourniquet, therefore, the meniscus will be in its posterior position, and if the knee is then merely allowed to flex to a right angle over the end of the table in the usual manner, unnecessary difficulty will be experienced at operation. In *Case 4*, for instance, the meniscus would have been found lying entirely behind the lateral femoral condyle. The last manoeuvre after applying the tourniquet in these cases should be to carry out full flexion of the joint and thereby snap the meniscus into its anterior position. The knee can then be allowed to extend from the fully flexed position to semiflexion, and the cartilage will be found in its most accessible position when the joint is opened.

SUMMARY

1. Four new cases and a new dissecting-room specimen of congenital lateral disc meniscus are described.
2. The manner in which the snapping knee of congenital disc meniscus is produced is discussed.

BIBLIOGRAPHY

(chronologically arranged)

Cases Described in Dissecting or Post-mortem Room.—

- 1889—YOUNG, R. B., in Cleland, Mackay, and Young's *Memoirs and Memoranda in Anatomy*, 179. London and Edinburgh: Williams and Norgate. (Three specimens.)
- 1895—HIGGINS, H., *four. Anat. and Physiol.*, 1895, xxix, 390. (Two specimens.)
- 1904—FICK, R., in Bardeleben's *Handbuch der Anatomie des Menschen*, ii, No. 1, 358. (One specimen.)
- 1911—POIRIER, P., *Traité d'Anatomie humaine*, 1911, i, 692. Paris. (Two specimens.)
- 1924—FISHER, A. G. TIMBRELL, *Internal Derangements of the Knee-joint, their Pathology and Treatment by Modern Methods*, 2nd ed., 1924, 12. London: H. K. Lewis & Co. (One specimen.)
- 1928—LANGER, M., *Anat. Anzeiger*, 1928, xlv, 409. (Two specimens.)
- 1931—FRÜND, Verein niederrein-Westfal. Chir. 1931, see *Zentralb. f. Chir.*, 1931, lviii, 2391. (This article refers to the condition, but does not describe a case. A personal communication regarding the case in question appears, however, in Jaroschy.)
- 1932—DUNCAN, D., *Anat. Rec.*, 1932, liii, 305. (One bilateral specimen from a negro.)

Clinical Cases Verified by Operation, with Detailed Description.—

- 1910—KROISS, F., *Beitr. z. klin. Chir.*, 1910, lxvi, 598. (One case with superadded tear.)
 1928—BRISTOW, W. R., *Proc. Roy. Soc. Med.*, 1928, xxi, 241. (One case.)
 1928—SCHULZ, A., *Arch. f. orthop. u. unfall Chir.*, 1928, xxvi, 315. (Two cases, one traumatic.)
 1932—ELLIS, V. H., *Lancet*, 1932, i, 1359. (Two cases.)
 1932—ROCYN-JONES, A., *Proc. Roy. Soc. Med.*, 1932, xxv, 395. (One case.)
 1933—LLOYD, E. I., *Lancet*, 1933, i, 525. (Two cases.)
 1934—FINDER, J. G., *Jour. Bone and Joint Surg.*, 1934, xvi, 804. (Three cases, one by personal communication from Timbrell Fisher; one traumatic.)
 1935—BELL-JONES, E., *Liverpool Med.-Chir. Jour.*, 1935, xliii, 78. (Collected fourteen new cases, eight of which were traumatic.)
 1935—JAROSCHY, W., *Beitr. z. klin. Chir.*, 1935, clxi, 139. (Complete survey of literature, comparative anatomy, and three new cases, one of which was traumatic.)
 1936—FISHER, A. G. TIMBRELL, *Brit. Med. Jour.*, 1936, i, 688. (Two cases, one previously described by Finder; one of these was traumatic.)

Clinical Cases Mentioned without Details.—

- 1930—FAIRBANK, H. A. T., *Proc. Roy. Soc. Med.*, 1930, xxiii, 1001, also *Brit. Med. Jour.*, 1930, i, 581. (Has operated on two cases.)
 1930—JONES, R. WATSON, *Proc. Roy. Soc. Med.*, 1930, xxiii, 1588. (Has seen two cases.)
 1934—DUNN, NAUGHTON, *Lancet*, 1934, i, 1267. (Has seen eight cases.)
 1936—MCFARLAND, *Brit. Med. Jour.*, 1936, i, 819. (Has seen nine cases.)

Other Articles Referred to.—

- 1927—STACKELBERG, VON, *Deut. Zeits. f. Chir.*, 1927, cciii, 204.
 1929—STACKELBERG, VON, *Ibid.*, 1929, ccxxi, 414. (Both these articles deal with a case of snapping knee in a five-months infant. In the second the pathological findings at post-mortem are shown, and do not warrant its inclusion as a case of disc cartilage.)

SOME EXAMPLES OF DISEASE OF THE VERTEBRAL COLUMN FOUND IN SKELETONS OF ANCIENT EGYPT A CONTRIBUTION TO PALÆOPATHOLOGY

BY L. R. SHORE, CAMBRIDGE

IN this paper it is proposed to offer descriptions of a few specimens which show different types of disease in the vertebral column. All the specimens to be described are contained in a collection of vertebral columns at Cambridge derived from excavations at Hierakonpolis, and are attributed to the Predynastic Epoch. The material includes about 4100 vertebræ, being the remains of 274 columns. The degree of damage or loss that has occurred will be appreciated from the statement that no more than eight columns are complete or capable of being reconstructed.

The term 'palæopathology' was coined by Sir Armand Ruffer to indicate that branch of scientific inquiry which is concerned with the pathological processes which affected the human body in the remote past. Obviously the material available for such inquiry is mainly limited to bones, except where exceptional circumstances and a high standard of the arts and crafts, as in Egypt, extended the field for research. Ruffer himself described the lesions of bilharzia, of arterial disease, and even of variola in tissues derived from mummies, in the collection of writings which have been published under the title *Studies in the Palæopathology of Egypt*. From dolls, bas-reliefs, and other sculptures Ruffer has given an account of dwarfs and deformities from which fairly exact diagnosis can be made. The same book contains a paper on "Arthritis Deformans and Spondylitis in Ancient Egypt". Having had a field for observation that ranged from archaic Nubians through Predynastic, Dynastic, and Ptolemaic Egyptians to the Greek and Roman periods and even to the Copts of early Christian times, Ruffer¹ formed an opinion that may well be stated in his own words:—

The pathological anatomy of spondylitis did not vary during this period of eight thousand years. Further, it is clear that geographical distribution did not influence the course or incidence of the disease, since specimens from Lower Egypt, Upper Egypt, and Nubia showed that the frequency, nature, and severity of the lesions were unaffected by varying climatic conditions.

The present paper is written in the hope of presenting some matter of interest though based upon nothing but dried and macerated bones. Probably no specimen will be described which might not be found in a modern skeleton in a modern museum of pathology. It is the presence of similar lesions in the vertebral columns of ancient Egyptians that constitutes their chief claim to interest.

All the specimens to be described have one feature in common, namely, ankylosis of certain members of the vertebral column. Closer examination of the specimens shows the presence of different causative processes bringing about this common effect.

It may perhaps be of service to recall in brief terms the more important mechanisms by which the units of the vertebral column are united in the natural state. Anterior to the spinal canal, the bodies of the vertebræ, by their cartilage-covered upper and lower surfaces, make contacts with the intervertebral discs, structures which possess central nuclei of a highly expansive nature restrained by layers of tough fibrous tissue. Short ligaments extending from vertebral body to vertebral body lie outside the intervertebral discs. Enclosing the vertebral bodies and the tissues between them around their circuit for the whole length of the column lies a fibrous envelope of which the well-known anterior and posterior common ligaments are specialized thickenings. Posterior to the spinal canal, the dorsal arches, by contact of each with its upper and lower neighbour, make a series of dorsal diarthrodial joints. In addition, the dorsal arches are connected by a series of powerful interlaminar ligaments (ligamenta subflava) and by the interspinous ligaments.

It follows, therefore, that when the perivertebral fibrous sheath is ossified a layer of new bone will be found to hide the normal superficial texture of the vertebral bodies. If sufficiently extensive, this new bone surrounds the site of the intervertebral disc, which is represented in a macerated specimen by an intervertebral space. When the deeper ligaments, those of no more than intervertebral length, are ossified, the normal appearance of the vertebral bodies is not lost at the bases of the osteophytes which are marginal in their place of origin.

The capsular ligaments of the dorsal intervertebral joints in an ossified state may enclose a joint either patent or obliterated by bony invasion; it is only by section that it can be determined which condition obtains.

Ossified interlaminar ligaments are recognized by their anatomical position.

SPECIMEN I.—ANKYLOSIS OF THE SECOND AND THIRD LUMBAR VERTEBRÆ

(Figs. 189, 190)

The anterior view in *Fig. 189* shows that the normal superficial texture of the vertebral bodies is lost. A superficial sheet of new bone, of a texture smoother than normal though porous here and there, somewhat raised and, as it were, *appliqué*, envelops the vertebral bodies and encloses the intervertebral space. It seems, therefore, that new bone has been laid down in the perivertebral fibrous sheath. On the right side this bony sheet is complete as far back as the intervertebral or neural foramen; on the left side and in front it is partly deficient and the normal surface of a vertebral body may be seen. A marked prominence over the intervertebral union projects towards the right side and makes the specimen asymmetrical.

The dorsal arches are not united: no ossific process has invaded the interlaminar ligaments, the capsular ligaments of the dorsal intervertebral joints, or the joint cavities themselves. *Fig. 190* is a photograph of a section made in a coronal plane and traversing the prominence on the right side. This prominence is shown to be a bridge of cancellous bone; the intervertebral space is not invaded; the lower (3rd lumbar) vertebral body is somewhat wedge-shaped, with the narrow edge towards the right.

It is certain that ankylosis is of long standing, for marrow spaces have penetrated the bony bridge of union from end to end. The texture of both bones is less compact than normal, and is certainly disorganized in the neighbourhood of the connecting bridge. Degeneration of the 3rd lumbar body is shown in its partial collapse into a wedge shape.

The agent which brought about the degenerative process probably was a microbic invasion—almost certainly blood-borne; otherwise it is difficult to account for the degenerative process being so generalized through the two vertebræ. One of the effects of this agent has been an intense stimulation towards new bone formation in the fibrous envelope of the two vertebræ.

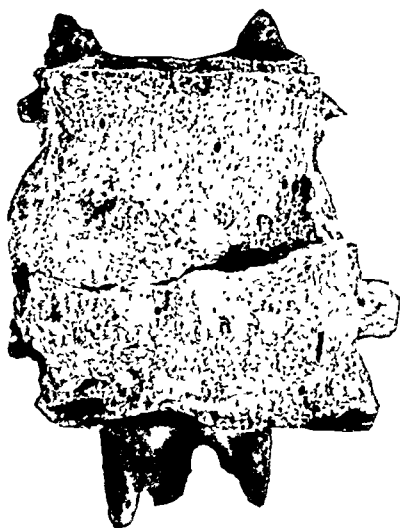


FIG. 189.

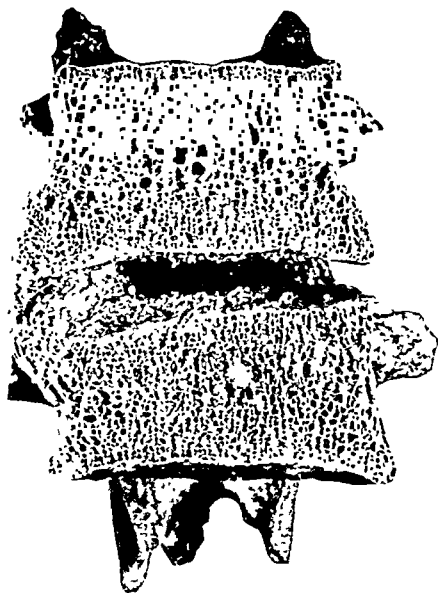


FIG. 190.

FIG. 189.—*Specimen I.* Ankylosis of the 2nd and 3rd lumbar vertebræ. The specimen is viewed from the front. A sheet of superficial bone, of texture smoother than normal, covers the two vertebral bodies and the intervertebral space except for a gap on the left front. A break in the superficial sheet extends from this gap towards the right; this break was the result of a mischance in handling the specimen, but it follows the intervertebral space. The new superficial bone corresponds to the superficial fibrous sheath of the vertebral column.

FIG. 190.—*Specimen I.* This photograph shows the appearance of a coronal section through the ankylosed 2nd and 3rd lumbar vertebræ. The intervertebral space is patent; the lower vertebral body is somewhat wedge-shaped, with the thin edge towards the right, and is also slightly hollowed. The texture of both bones is less compact than is normal. Cancellous bone is continuous in the bony bridge which connects the two vertebral bodies and corresponds to the prominence on the right side seen in the last figure.

The intervertebral space, so far from being reduced, is rather enlarged, for the lower vertebral body is slightly hollowed on its upper surface. This suggests that the normally expansive intervertebral disc met with decreasing resistance as the bone became softened by disease. Also it is shown that the intervertebral disc retained its turgor and therefore was not diseased. This conclusion goes far to negative the infecting organism as *Bacillus typhosus*, which, being generally credited with a predilection for the tissues of the intervertebral discs, leads to their early collapse in typhoid infections of the spine.

Except for arthritis this specimen goes far to reproduce the picture of 'spondylitis ossificans ligamentosa' described by Lawford Knaggs,² showing, as it does, superficial ossification of ligaments, preservation of the intervertebral space, and rarefaction of bone. Lawford Knaggs insists that these features, with arthritis, represent a vertebral manifestation of an infective or 'rheumatoid' process in the body elsewhere. Perhaps we are viewing a specimen which attained spontaneous cure, possibly after formation of the envelope of new bone which answered as a splint and secured immobility of the inflamed parts.

SPECIMENS II, III, AND IV.—ANKYLOSIS OF THE SECOND AND THIRD THORACIC, SIXTH AND SEVENTH THORACIC, AND THIRD AND FOURTH LUMBAR VERTEBRÆ

(Figs. 191, 192)

In one vertebral column bony ankylosis has occurred between three pairs of vertebræ, the 2nd and 3rd thoracic, the 6th and 7th thoracic, and the 3rd and 4th lumbar.

All three specimens are extraordinarily light to handle and very fragile; they have been so considerably damaged that certain of the superficial details are uncertain. The left lateral appearances of these three pairs of vertebræ are shown in *Fig. 191*.

The mechanism of union seems to be much the same for each pair of vertebræ. The dorsal arches are firmly united by bone and so also are the vertebral bodies. While it is easy to define the mechanism of union of the former, it is not at all easy to state by what means the vertebral bodies are joined.

In these specimens there is no obvious sheet of superficial bone such as was seen in *Specimen I*; it seems rather that vertebral body has formed direct union with vertebral body, because the vertebral bodies have an almost normal superficial texture.

Specimen II—2nd and 3rd Thoracic Vertebræ.—The surfaces of the two vertebral bodies show light superficial striation as is found normally. The position of the intervertebral joint is quite apparent, being marked by a ridge which bears upon it a transverse line with here and there a short linear gap. Part of the same gap is to be seen on both sides in the area for articulation of the head of the 3rd rib. This articular area, and the intervertebral interval, are shown in *Fig. 191*, ringed in white chalk upon the specimen before it was photographed.

From the side view it is evident that the vertical depth of the 2nd thoracic vertebra is much less than that of the 3rd, and the section in *Fig. 192* shows the same difference. That compression and distortion of the same vertebra have taken place is shown by two further findings in *Fig. 191*: first, the upper vertebral body bulges while the other is slightly hollowed; secondly, the 3rd costo-central joint is out of alinement with the 2nd and 4th, being situated in a more posterior plane.

Specimen III—6th and 7th Thoracic Vertebræ.—These two vertebræ are joined into a curve of slight anterior concavity, corresponding to the normal curve of the thoracic column. The surface of the two vertebral bodies is striated, scarcely different from the normal, but is continued below into a flange which projects about 2 mm.

The position of the intervertebral joint is indicated on the right side by a line about 4 mm. long, and on both sides by gaps in the areas for articulation with the

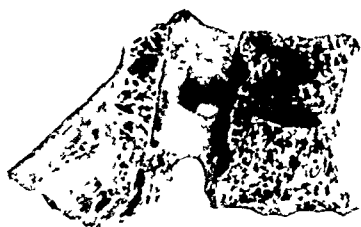
heads of the 7th ribs. The vertebral bodies are not hollowed, therefore no ridge marks the union, and the incidence of disease upon these two vertebræ does not seem to have been unequal. The transverse processes have been broken off, and so it is impossible to report on the condition of the costo-transverse joints. The three costo-central and dorsal intervertebral joints above and below the place of ankylosis show no signs of disease.



a



b



c

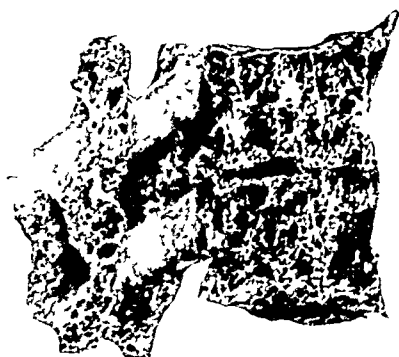


FIG 191

FIG 192

Specimen IV—3rd and 4th Lumbar Vertebræ.—*Fig. 191* shows that these two vertebræ have been considerably damaged; the transverse processes have been lost, and the ridge that covered the intervertebral union has been largely broken away.

In this specimen, near the upper and lower margins, the surface of the bone is smoother and less obviously striated than in the others, also it is somewhat bulky and punctured with small foramina. This appearance, together with its continuation into flanges of bone which project from each of the upper and lower edges of the specimen, suggests some ossification in superficial ligaments.

Both vertebral bodies are hollowed to a very considerable degree, and it seems that a conspicuous ridge, now broken away for the most part, must have marked their place of union. The dorsal end of this ridge partly occludes the intervertebral foramen. Where the intervertebral union has been exposed by superficial breakage no intervertebral space is to be seen—only bone of a very porous texture.

Fig. 192 shows photographs of the three pairs of vertebræ after section. In each specimen the plane of the cut through the vertebral bodies is true sagittal, but that through the dorsal arches passes to the right of the middle line.

FIG 191—Specimens II, III, and IV A photograph of three pairs of ankylosed vertebræ derived from the same column and viewed from the left side *a*, Th 2-3. *b*, Th 6-7, *c*, L 3-4

a Th 2-3 shows the articular surfaces, picked out in white chalk, for the heads of three ribs and for the 2nd costo-transverse joint, the 3rd costo-transverse joint has been lost with the 3rd transverse process. The 3rd costo-central joint shows a trace of the intervertebral space in the form of a narrow gap. This joint is in a plane posterior to that for the heads of the 2nd and 4th ribs, thus showing evidence of distortion. The 2nd vertebral body bulges somewhat, while the 3rd retains its normal slight hollow. The union of the two vertebral bodies is marked by a slight ridge, which bears traces of a line upon it. The general surface of the vertebral bodies is almost normal.

b Th 6-7 These two vertebræ are united in a curve which is nearly normal for this part of the vertebral column. The position of the intervertebral union is clearest in the area of articulation with the head of the 7th rib. No ridge marks the union, and the vertebral bodies are not distorted. Both transverse processes with their costo-transverse joints have been lost, but the costo-central and the dorsal joints above and below appear to be normal.

c L 3-4 These two vertebræ have been much damaged, having lost both transverse processes and the surface of the ridge that marked the union between the two bodies. Both vertebral bodies are hollowed, and the ridge that marked the union at the dorsal end considerably obstructs the intervertebral foramen. Where the ridge of union has been broken no intervertebral space is to be seen, only very porous bone. At the upper and the lower margins of the specimen flanges of bone project. These flanges are of smoother texture, and pierced by a few small foramina. The dorsal intervertebral joints above and below are normal.

FIG 192—Specimens II, III, and IV This photograph shows the three pairs of ankylosed vertebræ after section. The plane of the section through the vertebral bodies is true sagittal, that through the dorsal arches traverses the sites of the dorsal intervertebral joints. In all these specimens the dorsal joints have been completely obliterated, and cancellous bone occupies the former joint sites. Union of the dorsal arches is also effected by ossification of the interspinous ligaments, best seen in Th 6-7, and by ossification of the interlaminar ligaments, which is best seen in the upper and the lower specimens. The texture of the bones both in the vertebral bodies and in the dorsal arches is greatly rarefied. Compression of the 2nd thoracic body, previously remarked, is seen again. The intervertebral spaces are progressively invaded from the periphery, most conspicuously in the lowest and least in the highest of the three specimens.

The figure shows that in each specimen the vertebral bodies are joined across the intervertebral space—very extensively in the lowest, less so in the middle, and least in the uppermost pair.

Bony invasion of the intervertebral space probably commenced circumferentially and spread inwards into the intervertebral discs. In the 3rd and 4th lumbar vertebræ, the site of the most advanced and therefore the oldest pathological change, even the central parts of the intervertebral space have been invaded by new bone.

In each specimen the dorsal intervertebral joints are completely obliterated, and cancellous bone, i.e., marrow, is continuous from dorsal arch to dorsal arch across the former site of the joint. Not only the bone of the dorsal arches, but also the new bone which occupies the joint sites, is very porous. It is perhaps surprising that the costal joints, so far as they have been preserved, are normal.

The sections of the vertebral bodies show a texture which is very porous or spongy. It may be a matter of wonder that they are not hollow on their upper and lower opposed surfaces in response to the expansive pressure exercised by the intervertebral discs. The explanation probably lies in the loss of this expansive quality of the discs from disease, of which most ample evidence lies in their invasion by bone, the presumed effect of an inflammatory process spread from the vertebral bodies. That pressure was exerted by the intervertebral discs at some stage is shown by the section of the 2nd and 3rd thoracic vertebræ, which shows at once slight hollowing and the least degree of invasion of the disc. It follows, therefore, that softening of the vertebral bodies preceded bony invasion of the intervertebral discs.

The following are the outstanding features of *Specimens II, III, and IV*: ossification of the ligaments of the dorsal arches; arthritis of the dorsal intervertebral joints, evidenced by obliteration of the joint cavities; invasion of the intervertebral discs; and rarefaction of bone. Perhaps the processes which led to these results were something as follows.

Invading microbes, almost certainly blood-borne, settled in the bone of the vertebral bodies and of the dorsal arches, and in the dorsal intervertebral joints.

In the joints microbic activity led to destruction of cartilages and their substitution by granulation tissue. Fibrous adhesions prepared the way for osteogenetic cells to invade the former joint cavities from both of the articulating bones, and the result is osseous union by bone formation in fibrous adhesions. Bone formation in the interlaminar and the interspinous ligaments must have been initiated from the same source.

In the vertebral bodies microbic activity has manifested itself by bone destruction. Bony invasion of the intervertebral discs, as met with in these specimens, is somewhat uncommon, but must have spread from the vertebral bodies. *Specimen II* (Th. 2-3) shows that bone formation in the intervertebral disc is a later process than invasion of the vertebral bodies; direct spread is possibly delayed by the sheets of cartilage which intervene between the vertebral bodies and the intervertebral discs.

It might reasonably be held that the dorsal vertebral arches were the earliest site of infection. In his account of acute pyogenic osteomyelitis of the spine, Steindler³ expresses the view that the dorsal arches and the transverse processes are the commoner site of election for the pyogenic organisms, and points a contrast with the vertebral body which is preferred by the *Bacillus tuberculosis*. From time to time medical literature contains reports of cases which seem to resemble the three specimens here described. A recent report by Flemming⁴ contains a radiogram which presents an appearance very like *Specimen III*, and also situated in the thoracic column. In Flemming's case, and also in the opinion of Steindler, *Staphylococcus pyogenes aureus* is considered the causative organism. General opinion seems to be that typhoid has a predilection for the intervertebral discs and for the superficial investing ligaments, though rarefaction of bone may also occur. The deformity in these specimens is not of the kind to be expected from collapse of the intervertebral discs; indeed, we know that in *Specimen II*, at least, nothing of the sort occurred. It is unlikely that these three specimens are examples of the 'typhoid spine'.

SPECIMEN V.—ANKYLOSIS OF THE THIRD, FOURTH, AND FIFTH THORACIC VERTEBRÆ

(Figs. 193, 194)

This specimen consists of the 3rd, 4th, and 5th thoracic vertebræ ankylosed into a formation which, from the side view in *Fig. 193*, has an appearance very suggestive of Pott's disease on account of the ventral compression of the vertebral bodies and of the dorsal separation of the spinous processes.

Fig. 193 shows contact surfaces for the heads of four ribs on the vertebral bodies, and for the tubercles of three on the transverse processes. The fused mass is considerably hollowed, and the pedicle of the middle vertebral arch, projecting forwards, fails to make contact with the vertebral body, but ends in a blunt, rough stump.



FIG. 193.



FIG. 194.

FIG. 193.—*Specimen V.* In this figure is shown a photograph taken from the right side of the ankylosed 3rd, 4th, and 5th thoracic vertebræ. The vertebral bodies have assumed a wedge formation. The bodies are compressed together in front, while the spinous processes are splayed and separated to a corresponding degree. The pedicle of the middle vertebra is separated from the fused mass of the vertebral bodies in front. On the vertebral bodies and situated rather posteriorly can be seen four articular areas which correspond to the joints of four ribs which normally articulate with three thoracic vertebræ. The mass of vertebral bodies is distinctly hollowed, but there is no surface indication of the intervertebral spaces.

FIG. 194.—*Specimen V.* In this figure is shown a photograph of the right half of the specimen after sagittal section. It can be seen that the three dorsal arches are united by a continuous sheet of bone which corresponds to the ossified interlaminar ligaments and the ventral surfaces of the laminae. The separation of the middle dorsal arch from its body is seen in the stump of its pedicle. In the fused mass of vertebral bodies can be seen a space which is nearly central and elongated in the horizontal plane. This is the intervertebral space, which communicated with the exterior on the left side by a sinus. The bone above and below this space presents an orifice which comes to the surface in the neural canal. Each of these orifices contains the vena basis vertebræ and is a normal feature of a vertebral body. It seems, therefore, that two vertebral bodies lie on either side of the central gap, which must have contained the middle vertebra before it was broken down by disease and extruded through the sinus on the left side.

From the dorsum the outlines of the dorsal intervertebral joints are obscured by ossified capsular ligaments, but section has shown that the joint cavities are patent and the articular surfaces are normal. The interlaminar ligaments are extensively ossified.

In *Fig. 194* is shown the right half of the fused mass after sagittal section. The dorsal arches with the ossified interlaminar ligaments, as well as the stump of the

pedicle of the middle arch, can be seen. Anteriorly lies the mass of the vertebral bodies. In the middle lies a cavity elongated fore-and-aft and closed by a bridge of bone in front and behind. On either side of the gap lies a vertebral body which, though rather dense, seems very near to normal in the regular striation and in the presence of some part of a canal which communicates with the surface posteriorly. Such a canal in a fresh specimen transmits the vena basis vertebræ and is a normal feature of a sagittal section of a vertebral body. Therefore each canal indicates that a vertebral body does indeed lie on either side of the central gap. The central gap communicates with the exterior by a hole on the left side. Unfortunately it was not possible to obtain a satisfactory photograph of the specimen from the left side because it is too severely eroded and discoloured.

The inference is that the body of the third vertebra, which lay in the middle, has been destroyed by disease, and that two practically intact bodies are left above and below bearing between them on their dorsal arches that of the missing member.

Probably the 4th thoracic vertebra was destroyed by tuberculous disease, and extruded through the sinus on the left side. A slow process of destruction by an organism of low virulence rather than an acute osteomyelitis is probable. In this case the disease has been limited to a single vertebral body in a manner which contrasts with the specimens just described.

In this specimen the disease seems to have healed, to judge from secondary effects found in the column, and this again suggests tuberculosis.

These secondary or compensatory changes include extensive ossification of ligaments, particularly those of the dorsal joints and the interlaminar ligaments of the three fused dorsal arches. Further, no doubt as a response to the requirements of a new balance to the whole column, osteo-arthritic changes are present in the dorsal joints of the three vertebræ above and below the site of ankylosis.

SPECIMEN VI.—ANKYLOSIS OF THE SECOND AND THIRD LUMBAR VERTEBRÆ

(Figs. 195, 196.)

In a right anterior view, *Fig. 195* shows the two vertebral bodies united by an extensive sheet of smooth superficial bone which covers the bodies and the intervertebral space in an almost complete circuit, and therefore must correspond to the perivertebral fibrous sheath ossified. This sheet is complete except for one small gap in front and for a second on the right and behind, into which the pointer is inserted.

The dorsal intervertebral joints show some osteo-arthritis. The dorsal ligaments, except the interlaminar to a slight degree, are unossified. Sections of this specimen, shown in *Fig. 196*, display two cavities surrounded by bone and lying between the two vertebral bodies. One cavity is the intervertebral space, and extends fore-and-aft as far as the hinder and front surfaces of the vertebral bodies, where it is limited by ossified posterior and anterior ligaments. It was into this cavity that the pointer shown in *Fig. 195* was inserted. The other cavity is situated within the body of the 2nd lumbar vertebra, and is completely closed, being separated from the intervertebral space by a wall of thin but dense bone which can be seen best on the left-hand side in *Fig. 196*.

This cavity in the vertebral body is not quite central, being nearer to the posterior than to the anterior surface. It measures 1 cm. vertically and 1.7 cm. horizontally. The intervertebral space is encroached upon by the wall of the small

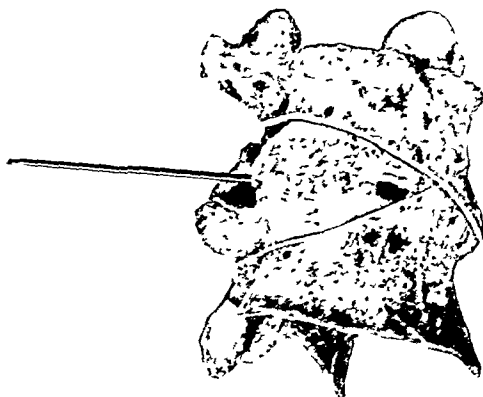


FIG 195—*Specimen VI* This photograph is a right anterior view of the 2nd and 3rd lumbar vertebrae united by a superficial sheet of smooth new bone. This new bone is complete except for the gap which can be seen in front and for a second gap in a right posterior position in which the pointer is inserted. The photograph was taken after section in a sagittal plane, and shows the line of section along the front of the specimen. The two halves are retained in position by two crossing rubber bands.

cavity, which is protruded almost to meet the other vertebral body. The interior is smooth and the wall is highly compact in texture; except for some increased density in the immediate vicinity of the cavity the texture of both bones is normal.

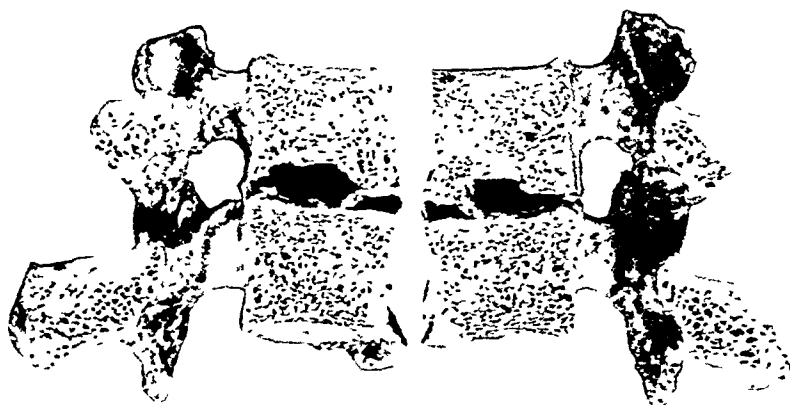


FIG 196—*Specimen VI*. This photograph presents the appearances after the fused vertebral bodies had been cut in the sagittal plane. It is to be noted that two cavities lie between the two vertebral bodies. One cavity—the larger and the lower—is limited by thin bone lying in the plane of the fronts and the backs of the vertebral bodies, and corresponds to the space formerly occupied by the intervertebral disc. A smaller cavity, rather oval in section, lies in the body of the 2nd lumbar vertebra and protrudes into the intervertebral space. This smaller cavity is completely enclosed in bone, and is shut off from the intervertebral space by a plate of thin but compact bone which is best seen on the left-hand side.

During life the small cavity must have contained a tissue which exerted pressure during its growth, for it formed a compact wall and encroached upon the space occupied by the normally expansive intervertebral disc. It is easy to surmise that

the compact new bone at each vertebral margin, where are attached the short intervertebral ligaments.

The nature of the pathological process which eroded the body of the 3rd lumbar vertebra and gave rise to these changes must be a matter of conjecture. An excavation of the size could scarcely have been caused by a meningeal tumour without erosion of the other vertebra. For the same reason a tumour arising in the intervertebral disc would scarcely explain the findings. A chondroma would have caused considerable distortion of the vertebral body in which it arose, and have left traces of expansion of the bone. It is perhaps most likely that we are examining the effects of tuberculous disease of the body of the 3rd lumbar vertebra.



FIG. 198—*Specimen VII* A photograph of the left half of the ankylosed 3rd and 4th lumbar vertebrae after sagittal section. There is an excavation in the posterior part of the body of the 3rd lumbar vertebra. The excavation is uniformly curved, and though the bone around it is somewhat condensed, the surface is porous and not compressed into a firm lamina as in *Specimen VI*. It can be seen that at the seat of ankylosis in front of the vertebral bodies, where the bridge of union broke in handling, the bone is very dense. From the two points of union compact bone spreads into the two vertebral bodies. The intervertebral space in front of the hollow excavation has parallel walls which expand just where the compact bone begins. It must be inferred that the intervertebral disc bulged anteriorly, and it is probable that the new bone is formed in the short intervertebral ligaments.

The presence of changes in the dorsal intervertebral joints elsewhere in the column suggests that there was time for the column to become adjusted to new strains, and therefore that the process, whatever it was, was not rapidly fatal. Osteo-arthritis in the dorsal joints is shown in the lateral drawing in *Fig. 197* affecting all the joints below the 10th thoracic. The interlaminar ligaments from the 9th to the 11th arches are ossified. The only part of the column available for reconstruction is shown in *Fig. 197*, from which it seems that the normal thoracolumbar curve has been straightened out and even reversed. There probably was a kyphotic curve to the whole vertebral column.

DISCUSSION

The specimens described illustrate the following diseases of the vertebral column to which ankylosis is a concomitant.

Specimen I.—Primary disease, probably microbic infection. Ankylosis is effected by ossification of the perivertebral fibrous sheath.

Specimens II, III, and IV.—Primary disease, probably staphylococcal infection. Ankylosis is brought about by obliteration of the joints, by ossification of the interlaminar ligaments and by bony invasion of the intervertebral discs.

Specimen V.—Tuberculous disease. Extrusion of a diseased vertebral body, and healing by ossification of ligaments.

Specimen VI.—Primary disease, a tumour of the vertebral body, probably a myeloma. Secondary ankylosis is brought about by ossification of the perivertebral fibrous sheath.

Specimen VII.—Primary disease uncertain, probably tuberculosis of the vertebral body. Ankylosis is caused by ossification of the short intervertebral ligaments.

In Ruffer's book are figured specimens which resemble *Specimens I to IV*, though the author does not subject them to very close analysis or to section. *Specimens I to IV* are all regarded as illustrating the effects of microbic infection, though these effects are much slighter in the first than in the others. Reasons for taking this view have been given already.

A feature common to these four specimens is rarefaction of bone. An obvious question arises: Is rarefaction of bone a sequel to ankylosis comparable with the atrophy of disuse? This question is not easily answered; but a limited experience, so far as it goes, does not suggest to me that rarefaction necessarily follows ankylosis. There is no rarefaction of bone in *Specimens V* and *VI*, though ankylosis is firm enough. It might be objected that there is no information available as to the relative chronicity of the pathological processes in *Specimens II, III, and IV* on the one hand, or in *V* and *VI* on the other. *Specimens V* and *VI* were ankylosed long enough for osteo-arthritic changes to develop in the dorsal intervertebral joints near the site of ankylosis in response, as it is surmised, to disturbances in balance of the whole column following on ankylosis.

In another place⁶ I have described a specimen of developmental sacro-iliac synostosis and compared it with synostosis of infective origin. The internal structure of the first was perfectly normal; it was in the second that alteration of bone structure with patches of rarefaction were found. It does not seem from this that ankylosis *per se* brings about absorption or rarefaction of bone; rather is this effect to be attributed to diffuse inflammation or osteomyelitis.

The infective nature of *Specimens II, III, and IV* would be very probable from the ankylosis and obliteration of the dorsal joints, apart from rarefaction of bone and invasion of the intervertebral discs.

The term 'rhizomelic spondylitis' has been used to include those cases of ankylosis brought about by an extensive superficial sheet of new bone. This term, derived from the Greek *ρίζα* spine, and *μέλος* membrane, is sufficiently descriptive of ossification of the perivertebral fibrous sheath. Rhizomelic spondylitis, as usually understood, corresponds to 'spondylitis ossificans ligamentosa' of Lawford Knaggs, and is distinguished by extensive ossification of the perivertebral sheath, but includes in addition ossification of the interlaminar ligaments, ankylosis of the small vertebral joints, and rarefaction of bone. An infective or 'rheumatoid' origin is the opinion almost universally held. There does not seem to be any justification for applying the term 'rhizomelic spondylitis' to *Specimens II, III, and IV*.

So far as external appearances go, *Specimen VI* might have been assigned to the category of rhizomelic spondylitis. Further examination shows that the

perivertebral ligaments are more probably ossified as the result of mechanical strain than as a consequence of 'rheumatoid' infection. A tumour encroached upon the intervertebral disc, which is normally expansive; hence excessive strain was set upon all of the ligaments which surround the disc and hold vertebral body to vertebral body, and that strain induced ossification in them.

That abnormal strain in ligament is sufficient cause for ossification is probably generally assumed, and the mechanism described below in the words of Macewen⁷ explaining the process of ossification in tendon probably represents a general principle which covers this theorem.

Tendons which are directly inserted into bone, without the intermediary of the periosteum, are, under exceptional circumstances, liable to osseous infiltration . . . mechanical irritation causes a proliferation of osteoblasts which penetrate the . . . fibres of the tendon and set up ossification in their midst. Some of the fibres . . . may retract into the tendon, carrying osteoblasts from their point of insertion. Rider's bone results in this way.

In another place⁸ I have tentatively explained the very common finding of ossified interlaminar ligaments as the result of strain or trauma following on ventriflexion of the vertebral column, and commonest in the thoracic region because the thoracic units are normally ventriflexed on each other and supported in that position by the interlaminar ligaments. This might be held to illustrate the principle set out above. 'Vertebræ with the interlaminar ligaments ossified to a greater or lesser extent are very common in Egyptian columns, and this makes the finding of Ruffer on this point the more remarkable. Writing on 'spondylitis deformans' in Egyptian skeletons, he states: "On the other hand the ligamenta subflava (interlaminar ligaments) composed of yellow elastic tissue always remained free from ossification".⁹ I strongly dissent from this opinion.

In *Specimen III* ankylosis is brought about by direct bony union of two vertebral bodies after extrusion of a diseased intermediate member. The interlaminar ligaments are completely ossified, it may be suggested, because they were subjected to strain in resisting separation of the dorsal arches in the position of extreme ventriflexion which followed collapse of the intermediate vertebral body. In the stages prior to healing, these ligaments must have been the chief local support of the three acutely flexed vertebræ, and probably represent the earliest site of new or reparative ossification.

In one of his papers Ruffer¹⁰ interprets two tomb carvings of the XI-XIIth and of the XVIIIth Dynasties as examples of Pott's disease of the spine, and states that he knows of no others in Egypt. His collection of bones apparently did not contain an example like *Specimen III*.

In several places reference has been made to concomitant osteo-arthritis in the dorsal intervertebral joints, and this finding has been adduced as evidence of chronicity. Timbrell Fisher defines osteo-arthritis as "the series of physiological or pathological changes that occur in a joint when it is subjected to oft-repeated injury, either mechanical or toxic, but of a moderate degree of intensity".¹¹ Pointing out that osteo-arthritis may be an expression of abnormal stresses laid upon a joint, Fisher gives among the causes of "traumatic or localized" osteo-arthritis this example: "a localized increase of articular stress of an occupational nature or caused by . . . ankylosis of an adjacent joint".

Ankylosis of two or more vertebræ may be supposed to cause an unusual stress to fall upon the dorsal intervertebral joints above and below the site of union,

and therefore to render those joints prone to the development of osteo-arthritis. This inference follows from Fisher's theorem, and also the further inference that in those specimens in which osteo-arthritis is an apparent concomitant of ankylosis the main pathological lesion must be of longer duration than is necessary to bring about the development of osteo-arthritis.

In his account of pathological lesions of the vertebral column Ruffer describes and figures specimens of ankylosis brought about, as it seems, by ossification of the perivertebral fibrous sheath, and a much larger number exhibiting osteophytes of the type that causes the familiar 'lipping' of the vertebral bodies. This type of osteophyte Ruffer considers to be that most characteristic of 'spondylitis'. Lipping, or polyspondylitis marginalis osteophytica, I believe to be the result of bone formation in the short intervertebral ligaments, brought about by strain when vertebra slips or rotates upon vertebra as a consequence of change in the intervertebral disc. Though this condition is the commonest of pathological changes in Egyptian vertebral columns, I have not obtained a specimen in which ankylosis had resulted except *Specimen VII*, and in this the initial lesion is highly unusual.

Ruffer makes an interesting deduction as to the development of the social sense in the communities of ancient Egypt. The individuals with the pathological lesions described in this paper must have been crippled and for a long time unable to exist by their own efforts, or to bear a share in the communal life of a primitive people. It is inferred that the care of the sick was a duty even in Predynastic times in ancient Egypt. *Specimen V*, healed tuberculosis of the spine, points in this direction, and even more strongly *Specimen VII* (if it is tuberculous), because diagnosis must have been much more difficult, and healing has taken place at a much earlier stage.

I have to acknowledge with thanks the opportunities given me by Prof. J. T. Wilson and by Dr. W. L. H. Duckworth to examine the series of vertebral columns stored in the Anatomy Department at Cambridge. Sir Walter Langdon-Brown, when Regius Professor of Physic at Cambridge, and the M.D. Committee, gave me permission to publish these observations, which formed part of a Thesis presented for the degree of M.D. in 1933.

REFERENCES.

- ¹ RUFFER, SIR MARC ARMAND, *Studies in the Palæopathology of Egypt*, 1921, 248.
- ² KNAGGS, R. LAWFORD, *The Inflammatory and Toxic Diseases of Bone*, 1926, 172 et seq.
- ³ STEINDLER, *Diseases and Deformities of the Spine and Thorax*, 1929, 463.
- ⁴ FLEMMING, CECIL, *Proc. Roy. Soc. Med.*, 1935, xxvii, 897, et seq.
- ⁵ SHORE, L. R., "Polyspondylitis Marginalis Osteophytica", *Brit. Jour. Surg.*, 1935, xxii, 850.
- ⁶ SHORE, L. R., "A Specimen of Developmental Sacro-iliac Synostosis", *Jour. Anat.*, 1930, lxi, 503.
- ⁷ MACEWEN, WILLIAM, *The Growth of Bone*, 1912, 135.
- ⁸ SHORE, L. R., "A Report on the Nature of Certain Bony Spurs Arising from the Dorsal Arches of the Thoracic Vertebrae", *Jour. Anat.*, 1931, lxxv, 379.
- ⁹ RUFFER, *Loc. cit.*, 242.
- ¹⁰ RUFFER, "On Dwarfs and other Deformed Persons in Ancient Egypt", *Loc. cit.*, 42.
- ¹¹ FISHER, TIMBRELL, *Chronic (Non-tuberculous) Arthritis*, 1929, 89 et seq.

MULTIPLE ARTHRITIS IN PRESUMABLY TUBERCULOUS SUBJECTS: DIFFICULTIES IN DIAGNOSIS AND TREATMENT

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INTRODUCTION

Diagnostic Inaccuracies.—Tuberculosis of a single large joint, e.g., hip or knee, in young persons before the age of puberty, can usually be diagnosed correctly after clinical and radiological examination. After puberty other types of 'non-specific' infective arthritis may make their appearance, and these must be considered in arriving at a diagnosis. Smith and Watters¹ found that the diagnosis was wrong in 22 per cent of 208 patients admitted for tuberculosis of the hip to the County Branch of the New York Orthopædic Hospital, and Smith² reports the findings in 24 cases of monarticular arthritis simulating tuberculosis which were submitted to exploratory operation. Histological examination and animal inoculation excluded tuberculosis in these cases. Smith remarks that the classical signs of tuberculosis of a joint are just as common in other joint diseases. Milgram³ points out that diagnostic inaccuracies may operate also in the other direction, and that 38·7 per cent of 142 proved cases of tuberculosis of bone, joint, and bursa were not so diagnosed. Ghormley and Brav⁴ record errors of diagnosis in 24·4 per cent of cases of supposed tuberculous arthritis and 13·7 per cent of supposed non-tuberculous arthritis affecting the knee.

Diagnostic Methods.—

Guinea-pig Inoculation of Aspirated Material, and Histological Examination of Tissue obtained by Biopsy.—The necessity of these methods of diagnosis is repeatedly stressed in American literature. The consistently obvious demonstration of tubercle formation in Allison and Ghormley's⁵ cases convince one of the value of the histological method, and Ghormley and Brav⁴ found the histological examination to be correct in all but 3·2 per cent of 158 cases, although in 22 cases additional sections were necessary in order to demonstrate the lesion. Out of 24 tuberculous cases tested by animal inoculation, 3 failed to demonstrate the nature of the disease. Biopsy is not so commonly practised in Great Britain, and Girdlestone⁶ writes that diagnostic arthrotomies are not always indicated in suspected tuberculosis of the knee, and false negatives are said to be common: 'a view which is also held by Forrester-Brown⁷ in regard to the early diagnosis of hip-joint tuberculosis.

X-ray Diagnosis.—The question of the radiological diagnosis of early joint tuberculosis has been widely discussed. Although Fraser,⁸ Rogers,⁹ and other authors believe that the primary tuberculous focus is more often in the synovial tissues of a joint than in the bone, Allison and Ghormley⁵ have found coincident

invasion of subchondral bone in all their cases. These authors make it clear, however, that it is possible to have active tuberculosis in bone and joint without any demonstrable change in the X-ray picture.

Smith¹⁰ says that decrease in the width of the joint space is a fairly early sign, and that evidence of bone destruction was present in very few of the early cases. Phemister^{11, 12} has shown that in tuberculous arthritis, in contradistinction to other forms of infective arthritis, the articular cartilage is often preserved at the points of contact of articular surfaces, and in radiographs the cartilage space often maintains its usual width.¹³ Certainly, if the joint space is maintained, in spite of associated bone destruction, there is a strong presumption of tuberculosis (Pomeranz),¹⁴ but loss of joint space is not a point against tuberculosis. Radiology does not help in the differential diagnosis until the comparatively late stages when there is evidence of bone destruction.

Henderson and Fortin¹⁵ think there is no typical X-ray picture of tuberculosis of the knee. They rely on the history and objective data to reach a diagnosis, and in differentiating tuberculosis from 'chronic infectious arthritis' they note that the latter is commoner in more elderly patients, and that in non-specific arthritis of the knee-joint either other joints become involved within a year, or the knee recovers after suitable treatment such as eradication of infective foci. In our experience, however, neither of these results may ensue, and the diagnosis may still remain in doubt. In this connection it may be well to recall the dictum of Smith¹⁰: "The idea that it is safer to treat the case conservatively at first and to await developments is a fallacious one, because, once immobilization is started, the condition is obscured indefinitely." Smith confines his cases to bed, but without immobilization, for such a time as may allow an evanescent arthritis to clear. If this does not occur, he performs an exploratory operation.

Tuberculin Reaction.—Without indulging in a full discussion of this well-worn subject, we may say with Atsatt¹⁵ that a positive reaction, quantitatively controlled, can only be presumptive of osseo-articular tuberculosis until all other possible active foci have been excluded, whereas a negative reaction, in the absence of certain modifying factors (overwhelming tuberculous infection, advanced sepsis, anæmia, or other grave disease), can eliminate the diagnosis of tuberculosis with some certainty. Occasionally an acute focal reaction in a joint, as in the case described by Bezançon and his colleagues,¹⁷ may indicate the tuberculous nature of a local arthritis. Reliable focal reactions are, however, rarely obtained when using the intracutaneous or Mantoux technique. We should like to call attention to the Tuberculin P.P.D. (Purified Protein Derivative) manufactured by Parke, Davis & Co., with the small first dose of which all highly sensitive cases can be detected without danger of a very severe reaction (Long, Seibert, and Aronson¹⁸). This is an accurately standardized preparation, with which quantitative reactions of some value can be obtained.

Considerable difficulties, therefore, may exist in the early differential diagnosis of the monarticular arthritides in adolescence and in the first decade of adult life. This matter of diagnosis is of far more than theoretical importance. Early immobilization might prevent destruction of bone in a tuberculous joint, but in a non-specific arthritis would not only subject the patient to an unnecessarily long and expensive in-patient treatment, but might result in an otherwise preventable

ankylosis. A joint with only 30° of useful movement is a far more valuable possession than an ankylosed joint, no matter how perfect the orthopædic position may be.

MULTIPLE ARTHRITIS AND TUBERCULOSIS

It may not seem so likely that confusion should occur in diagnosis in the case of a multiple arthritis, but that this does happen has been made clear in our experience and in the case records which we present. Many factors account for this confusion :—

1. The insidious monarticular onset of some cases of multiple non-specific arthritis.
2. The coexistence in the patient of some visceral tuberculous lesion, which may or may not influence the course of a non-tuberculous polyarthritis.
3. The possible occurrence of a single tuberculous joint superimposed upon a non-tuberculous multiple arthritis.
4. The occasional incidence of true tuberculous arthritis in two or more joints.
5. The comparative infrequency of non-specific arthritis of the hip in patients under middle age, and the tendency to suppose such a condition to be tuberculous.
6. Modification of the course of non-specific arthritis due to early immobilization.
7. The possibility that there exists an atypical tuberculous form of polyarthritis—tuberculous rheumatism.

We need not again emphasize the importance, both for treatment and prognosis, of a proper knowledge of these questions in diagnosis. Each will be discussed and illustrated, where possible, by cases from our own experience.

1. The Insidious Monarticular Onset of some cases of Multiple Non-specific Arthritis

Case 1.—Female, employed as machinist. No family history of tuberculosis. In 1929 she developed pain and swelling in the right knee. Three months after onset this was diagnosed as tuberculous arthritis of the knee. She was sent to a sanatorium and treated in extension and later in plaster for $3\frac{1}{2}$ years. At the age of 23, while still in the sanatorium, symptoms arose in the left elbow and in the left wrist. These were still thought to be tuberculous, and were treated by fixation in plaster. The wrist became ankylosed, with an awkward radial deflection of the hand and wrist-drop. The symptoms in these three joints gradually subsided and she returned home in 1933.

A year later, however, an arthritis developed in the right elbow, and on admission to the Harrogate Royal Bath Hospital at the end of 1935, this was the most actively diseased joint. Her condition after admission to this hospital was as follows :—*Rt. elbow* : Fluid swelling of joint ; the muscles supporting the joint were extremely atrophied and weak ; almost full passive movement could be obtained. *Lt. elbow* : Flexion and extension limited to about 30° of movement ; no pain. *Lt. wrist* : Rigid, with radial deviation of the hand. *Rt. knee* : Full extension not obtainable, but about 70° of movement possible. X-ray examination showed slight erosions and loss of cartilage space in all joints, bony ankylosis of the left wrist, and secondary osteophyte formation and periosteal reaction in right knee and left elbow. Aspiration of fluid from the right elbow was performed on two occasions. The total nucleated cells numbered 34,500 and 40,000 per cm., with 90 per cent of polymorphs (commensurate with that found in the rheumatoid type of chronic arthritis¹⁹). Cultures were sterile, and guinea-pig inoculation negative. The anterior end of the 4th right rib was splayed out as if by medullary expansion. There was a slightly tender swelling over this site at the time of her admission, but the swelling subsided. We are unfamiliar

with the radiographical appearances: they were not suggestive of tuberculosis. Blood Wassermann reaction was negative on two tests. Tuberculin reaction with P.P.D. Tuberculin was 3 + with the first test dose; no active focus of tuberculous infection, however, was found.

Physical therapy and general spa treatment greatly improved this patient, restoring almost full active movement to the left elbow and a bigger range of movement in the right knee. She has since returned to her work.

This is an unusual form of multiple arthritis to encounter in a female. The small joints of the hands and feet were never involved and the lesions were asymmetrical. Against the original diagnosis of tuberculosis were the absence of definite radiological evidence of tuberculosis even after six years of the disease, the particularly chronic course of the disease, the examination of the joint fluid from the elbows, and the improvement of function resulting from methods of physical therapy. The only confusing point was the strongly positive tuberculin reaction. The possibility of syphilis was considered, but discarded after failing to find any clinical confirmation, and after two negative Wassermann serum tests. No source of infection was discovered. Periods of one or two years elapsed between the successive joint involvements. There is no doubt that this slow progress of the disease accounted for the confusion in diagnosis. It is impossible to say to what extent the immobilization and general régime of the sanatorium influenced the course of the disease, and delayed the spread to other joints. Apart from the possibility that the condition might have become more generalized had she not been so dealt with, we think that the treatment adopted was detrimental to functional activity of her joints, and that the period of her economic incapacity was unduly prolonged.

2. The Existence of some Visceral Tuberculous Lesion which may or may not Influence the Course of a Non-tuberculous Polyarthrititis

Case 2.—Mrs. A. E., aged 59 years, was seen by one of us (C. C.) at the request of her doctor, and was admitted to East Fortune Sanatorium. We had known for many years that she suffered from rheumatoid arthritis, but the reason for the requested visit was a sudden hæmoptysis.

She had evidence of old tuberculous disease in the form of neck glandular scars, and chains of small very hard glands were easily felt in both posterior triangles of the neck. When questioned, she gave a history of a glandular abscess at the age of 16 years, and of the removal of her right index finger for disease which appeared at the age of 24 years after a trivial accident. This was almost certainly a tuberculous condition. The X-ray examination showed the shadows of densely calcified glands in the roots of both lungs, small calcified shadows below both clavicles, and a light infiltration, which appeared to be of recent development, of the upper half of the left lung. She had advanced rheumatoid arthritis, with the typical deformities of the advanced disease. Every joint in her limbs appeared to be affected, and the deformities of hands and feet were gross. The disease was most active in the knee-joints, each of which had a flexion deformity and subluxation of the tibia with genu valgum. The X-ray appearances were typical of advanced rheumatoid arthritis. She was a thin woman of poor physique, and there was gross muscle atrophy associated with the joint conditions. She had a good deal of intestinal discomfort, and her stools were bulky and semi-formed, intensely fetid, and contained undigested food. Fractional gastric analysis showed a complete achylia. She improved very greatly with treatment in the Sanatorium. This treatment included two courses of gold injections, a long course of careful orthopædic and physical treatment, and finally a double osteotomy to correct the gross bilateral genu valgum.

Case 3.—Mrs. A. A., aged 38, was admitted to East Fortune Sanatorium on Oct. 12, 1929. She gave a history of having suffered from pleurisy 18 years previously, and of having had intermittent fever and cough since a confinement 12 years before. Four years after the

development of the cough she developed rheumatoid arthritis, and when she came to the Sanatorium was in a very poor condition. Her sputum contained tubercle bacilli, and the X-ray film showed dense fibroid tuberculous disease of the left upper lobe containing several cavities, with lighter fibrotic disease of the lung below and of the right apex. She had frequency of micturition and pain, with intermittent pyuria, which we finally traced to the presence of stones in the bladder and right kidney. On March 28, 1932, Mr. Mercer, surgeon to the Sanatorium, removed a stone weighing 19 gr. from the pelvis of the right kidney, and on May 5 he removed a mulberry calculus of the size of a horse-chestnut from the bladder. The urinary symptoms did not recur. She had extensive rheumatoid changes in elbows, wrists, knees, feet, and in the small joints of the hands, with characteristic X-ray appearances. Some years previously her tonsils had been removed and an infected left antrum had been drained. If one were to look for an initial focus, the urinary and other sepsis would have to be considered in addition to the tuberculous disease of her lungs.

The lung disease improved greatly, contraction of the fibrotic left upper lobe being helped by a phrenic evulsion which was performed on June 25, 1931. The joint condition had begun to improve before the kidney and bladder operations, and it seemed to us to improve coincidentally with the improvement of her lung disease. At the time of her discharge the disease in most of the joints was quiescent, and though many of them were deformed they had a useful range of painless movement. The left knee-joint remained swollen and rather painful, but was improving.

It is difficult to assess the part which tuberculosis may have played in the rheumatoid arthritis of these two patients. In both cases the tuberculous infection preceded the appearance of the rheumatoid condition, and it would be an easy conclusion to state that the one was secondary to the other. Such a view cannot be readily accepted, for in both patients there were many other points to consider. In *Case 3* there was a history of sepsis antecedent to the rheumatoid arthritis, while in *Case 2* there were intestinal disturbances (achylia and foetid stools) which might with equal reason be held as relative to the rheumatoid disease. We are inclined to regard the tuberculous disease and the rheumatoid arthritis as independent affections in these two patients.

The two diseases occur but rarely in the one patient. These two patients (*Cases 2 and 3*) are the only cases of true rheumatoid polyarthritis which have been admitted to East Fortune Sanatorium during the past 13 years. In that time 1562 patients over the age of 15 years with pulmonary tuberculosis, and 617 patients over the age of 15 years with non-pulmonary tuberculosis (207 of whom suffered from bone or joint disease) have been treated at the Sanatorium. This incidence is slightly lower than that found by Lawrason Brown at Saranac Lake (quoted by Pemberton²⁰), whose figures were 11 cases of positive rheumatoid involvement among 4499 cases of pulmonary tuberculosis. Pemberton states that active tuberculosis was very infrequent among patients with non-specific arthritis, and Brav and Hench²¹ discovered only 8 out of 250 patients with rheumatoid arthritis who had definite tuberculosis elsewhere than in the joints—a lower incidence of tuberculosis than in a corresponding number of non-arthritic patients. It is clear, then, that tuberculosis is sufficiently rare in rheumatoid arthritis, and polyarthritis is sufficiently rare in tuberculosis, to enable us to refute the suggestion that tuberculosis is a frequent or an important factor in the etiology of the other disease. The occasional coincidence of the two diseases in the one patient is illustrated by our two cases and by a few others in the literature, but we intend to postpone the more detailed discussion of these and of what is meant by 'tuberculous rheumatism' until we have described our other cases and the different problems which they present.

3. The Occasional Occurrence of a Single Tuberculous Joint Superimposed upon a Non-tuberculous Polyarthritis

Pathological observations by Brav and Hench²¹ disclosed evidence of tuberculosis of a single joint in 8 out of 75 cases diagnosed before operation as chronic infective polyarthritis. In each of these 8 cases the diagnosis of tuberculous arthritis of the single joint was suspected before operation, but the associated generalized arthritis introduced a confusing factor. While Brav and Hench cannot entirely disregard the possibility of an atypical multiple tuberculous arthritis, they suggest that tuberculous infection may have developed in one joint, the resistance of which was lowered by previous attacks of non-tuberculous polyarthritis. This seems especially probable in five of their patients in whom the generalized arthritis was primary, localization occurring later in one of the joints. Our *Case 4* is an illustration of this type. A severe arthritis of one knee, clinically and radiographically suggestive of tuberculosis, developed some years after the onset of a generalized arthritis in a young girl. A marked focal reaction in this knee and a general reaction followed the injection of tuberculin. There was also a tuberculous focus in the lung.

Case 4.—J. P., female, aged 16 years. At the age of 8 years, this patient had an attack of rheumatism with stiffness and pain in both wrist-joints. The attack lasted about seven months. At the age of 11, and again at 14 years, she had further attacks of rheumatism, the disease spreading to, and remaining in, the elbows, shoulders, knees, feet, and hands. There was no cardiac disease.

On admission to the Harrogate Royal Bath Hospital, she had all the appearances of chronic non-tuberculous polyarthritis. The wrists had a few degrees of movement, although radiographically they showed bone atrophy with scattered centres of rarefaction and slight erosions. The elbows were swollen and not very painful, and showed no significant changes on X-ray examination. The shoulders and ankles were stiff and limited in movement. There was spindle deformity of many of the finger-joints. The right knee appeared to be unaffected. The left knee, however, was swollen, hot, and extremely painful to touch or movement. Starting pains at night were experienced in this joint. There was muscle wasting above the joint. The X-ray film showed slight bone rarefaction and erosion in the region of the tibial spines, the cartilage space being well maintained. The affection of this (left) knee had developed within the last few months, and it was clear from the history she gave that the joint had been becoming progressively worse, while the other joints had been improving. An intradermal tuberculin test was made with 0.1 c.c. of 1-1000 Old Tuberculin, and a +++ local reaction developed. At 48 hours after the test the temperature had risen to 99.6°, there was general malaise, and a very marked exacerbation of symptoms in the left knee. No focal reaction occurred in any other joint. There was clinical and radiological evidence of a not very active tuberculous focus in the right infra-clavicular area of the lung.

The left knee was diagnosed as true tuberculous arthritis, and was immobilized in plaster. The patient was later transferred to an orthopaedic hospital.

4. The Occasional Incidence of True Tuberculous Arthritis in Two or More Joints

In a series of 168 cases of tuberculous arthritis of the knee, Ghormley and Brav⁴ found two joints affected in 13.1 per cent, and more than two joints affected in 5.4 per cent, of their cases. Brav and Hench,²¹ however, consider that when more than three joints are the seat of arthritis it is not very likely that the arthritis is due to tuberculosis. Allison and Ghormley⁵ describe one proved case of multiple tuberculous arthritis affecting elbows, wrists, knees, and tarsus. In this case there

was much bone destruction and periosteal reaction, also general glandular enlargement, and discharging sinuses leading from the right tarsus. This case is so clearly one of multiple tuberculous foci, that it in no way resembles the much debated form of 'tuberculous rheumatism'. We believe that the infrequency of multiple tuberculous arthritis has been exaggerated in common medical opinion. In East Fortune Sanatorium, during the past thirteen years, there have been at least 7 patients with multiple foci among 207 cases of osseo-articular tuberculosis. From these we may quote *Cases 5* and *6* as examples of the condition. This type of disease must be considered when reaching a diagnosis in an atypical multiple arthritis, but evidence of the true tuberculous nature of the condition is nearly always readily at hand.

Case 5.—J. T., female, aged 24 years, was a patient in East Fortune Sanatorium from 1930 to 1934. At the age of 18 she had suffered from pleurisy, which was followed by a rib abscess. At the age of 24, following an injury, her right knee became swollen, and it was for this condition that she was sent to the Sanatorium.

There was a typical tuberculous arthritis of the right knee, and the joint was finally excised and fixed by arthrodesis. Previously unsuspected tuberculous disease of the dorsal spine was found on admission. This involved the 5th, 6th, and 7th dorsal vertebrae. These lesions healed well, but she died two years later of tuberculous meningitis.

Case 6.—S. McG., female, aged 30 years, was admitted to East Fortune Sanatorium on June 18, 1932, and died on July 12. She had tuberculosis of her lungs with tubercle bacilli in her sputum. X-ray examination showed a large calcified abscess overlying the upper part of the sacrum and obscuring the lower lumbar spine. She had had spinal symptoms some years previously, and there is no doubt that this was an old psoas abscess. There was advanced tuberculous arthritis of the left knee-joint, which was full of tuberculous pus. The right hip-joint was stiff and painful, and the X-ray film showed extensive destruction of the joint with ankylosis.

5. The Comparative Infrequency of Non-specific Arthritis of the Hip in Patients under Middle Age, and the Tendency to Suppose the Condition in such Persons to be Tuberculous

Symptoms in five of the patients whose notes are included in this paper commenced in the hip-joint. These patients were diagnosed, in the first place, as suffering from tuberculous hip disease. The subsequent course of the disease and later investigations rendered this diagnosis untenable. Two of them we believe to have been cases of ankylosing spondylitis, the typical course of which was modified by their early treatment, and these will be described in the next section. Three others (*Cases 7, 8, and 9*) suffered from an atypical type of arthritis of the hip-joints. None resembled the usual form of rheumatoid arthritis, for the smaller peripheral joints were not implicated.

In our own experience objective or subjective signs of hip-joint disease are exceedingly uncommon in rheumatoid arthritis. Pemberton²⁰ also says that in the hip external visible evidence of the true pathological process may be lacking throughout, and Forestier²² states that he found only 10 cases of affection of the hip in more than 250 cases of progressive polyarthritis, and only 3 of these were bilateral. In only one instance in Forestier's cases did symptoms in the hip initiate the disease. Forrester-Brown²³ discusses the differential diagnosis of abnormal hip conditions in children, and mentions "arthritis of true rheumatic origin, less acute than

rheumatic fever, and another of quite unknown origin" as common causes of hip symptoms in children. She considers that the majority of so-called tuberculous hips which healed in the past with little or no immobilization were of this nature, and Telford²⁴ thinks that recovery with full movement of a hip-joint, diagnosed as tuberculous, must cast considerable doubt on this diagnosis.

We wish to emphasize, however, that though a painless joint will be the eventual result in all cases of non-tuberculous arthritis, immobilization, adopted on an incorrect diagnosis, may result in serious and unnecessary limitation of movement and may also actually delay recovery.

Case 7.—J. W., female, was admitted to East Fortune Sanatorium on Nov. 12, 1930, with a diagnosis of tuberculous disease of the left hip-joint. She was then 13½ years of age, and had been quite well until a month previously when pain appeared in this joint. The onset of the pain was gradual. She was admitted to a general hospital where it was observed that a dull pain was constantly present and referred to the outer aspect of the thigh, the front of the knee, and the calf. A few hard glands in the right posterior triangle of the neck had been detected, and despite somewhat indefinite X-ray findings she was diagnosed as having tuberculosis of the left hip-joint.



FIG. 199 —*Case 7*, Aug. 19, 1932. Non-specific infective type of arthritis of both hip-joints. The left hip has been affected for 18 months, the right for about 4 months. There is loss of cartilage space of both joints and general loss of bone density, more marked on the left side. In spite of the duration of the affection there is little or no erosion of bone, and recovery with greatly increased movement was eventually obtained.

On admission to the Sanatorium the patient was in a poor general condition, with a dry skin and appearances which suggested a chronic tuberculous infection. She gave a strongly + reaction to the Mantoux test (1-1000), and the left leg was fixed in a position of slight abduction and external rotation. There was tenderness to pressure on the head of the femur, all hip movements were in abeyance, and many small hard glands were present in the left inguinal region. An X-ray examination of the left hip showed merely loss of bone density and slight narrowing of the joint space, and, though not definitely diagnostic of tuberculous disease, that diagnosis was accepted.

A few days after admission extension was applied to the limb. By February, 1931, movement had returned to this joint, but the left knee was now enlarged and its range of movement was diminished. Her general condition was much better. By November, 1931, movement of the left hip had again become restricted, the left knee was very stiff and painful, and creaking and stiffness had appeared in the right knee. During this time the diagnosis of tuberculosis had been questioned on several occasions, but the diagnosis was not altered.

After symptoms had appeared in the right hip a radiograph showed narrowing of the joint space of both hips, with loss of bone density, more marked on the left than on the

right side. The contour of the articular surfaces remained regular in spite of the disease having been present in one of the joints for 18 months (*Fig. 199*). This was in April, 1932, and extension was applied to both legs. Movement became gradually less until at the beginning of 1934 the left hip seemed to be ankylosed, both knees were almost completely stiff, and movements of the right hip were reduced to a few degrees of flexion. Under a general anæsthetic, however, both hips moved freely and movement was present in both knees. The diagnosis of tuberculosis was then definitely discarded. Other lines of treatment were adopted to deal with the multiple arthritis. These included a course of T.A.B. injections, massage, and passive movement of the joints, and some improvement was effected.

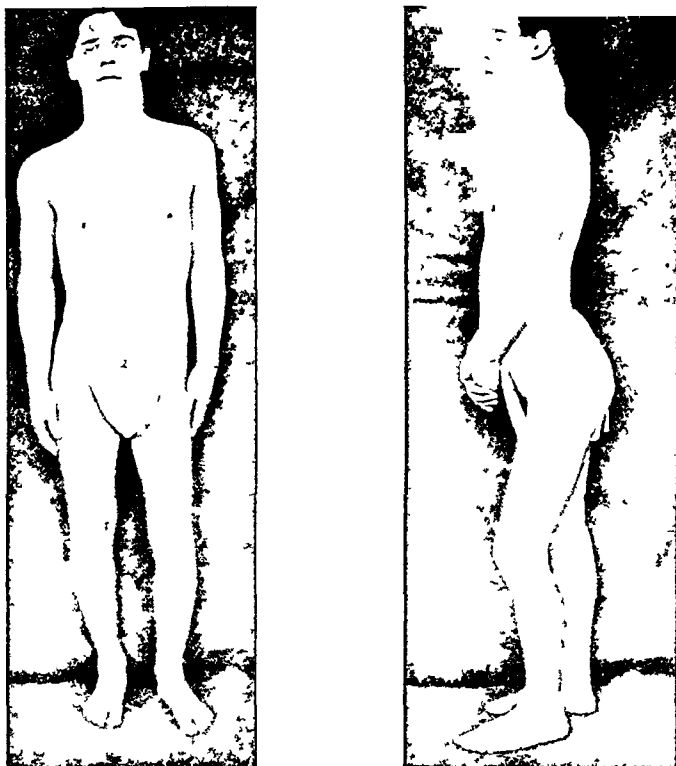
On Aug. 27, 1934, the patient was transferred to the Harrogate Royal Bath Hospital. She had still extreme limitation of movement of both hips, and wasting of the thigh and gluteal muscles. Her knees were stiff and crepitus was present in them, but there were several degrees of active movement. Pain was not conspicuous, but fatigue resulted from very little exercise. She walked with great difficulty with two sticks. The hips were held rigidly, and progress of a shuffling type was possible only by swinging the whole body. Treatment consisted of sulphur immersion baths and H.F. diathermy to both hips, followed by gentle massage and movements. She recovered movement and strength in a remarkable manner. After five weeks she could walk a distance of more than a mile, only using one stick when going uphill. Movement of the hips continued to increase and she was discharged on Oct. 13, 1934. Her condition has continued to improve, and she now leads a life of normal activity.

The case of this patient illustrates very clearly the type of arthritis of the hip in young persons in which diagnosis is mistaken and treatment misapplied. It is possible that the condition is far commoner than is generally recognized, and that many young patients have suffered unnecessary years of immobilization. Our patient was a tuberculous subject, but there is no reason to think that the arthritis was tuberculous, and we see now that the prolonged rest and extension was incorrect. Immobilization in her case only resulted in further limitation of movement of the joints, but function was fortunately regained after a course of spa treatment. All the early appearances of this patient were suggestive of tuberculous disease. The diagnosis was finally corrected by the absence of radiological evidence of bone destruction even after some years, the development of multiple joint affections, and the freedom of joint movement under anæsthesia.

Case 8.—A. B., male, aged 14 years, was admitted to East Fortune Sanatorium on May 7, 1930. In March of that year he had developed slight pain in the left hip. He was seen on several occasions as a surgical out-patient at a general hospital, and was diagnosed as early tuberculous disease of the joint.

He came into the Sanatorium in poor general condition. His tonsils were enlarged and inflamed and he had many carious tooth stumps. Chains of small hard glands were present in the posterior triangles of both sides of the neck, and he gave a strongly positive reaction to the von Pirquet test. The left hip-joint was in a normal position, but movement was restricted in all directions. X-ray examination of this joint was inconclusive; there was loss of bone density around the joint and a slight irregularity of the lower third of the articular surface of the femoral head. The limb was fixed in plaster for a period of eight months. At the end of that time he had painless movement of the hip-joint in all directions to about 50 per cent of the normal range. The X-ray film then showed narrowing of the joint space, but no further bone destruction. The diagnosis of tuberculosis was doubted and the patient was now left in bed without plaster and encouraged to move the joint. Stiffening, however, took place, and he left the sanatorium with only a few degrees of movement of the hip. During his stay he had several crops of subcutaneous abscesses, possibly related to his dental infection.

The patient was seen at intervals under the After-Care Scheme, and on Nov. 5, 1935, it was found that the left hip had become fixed in a position of flexion and slight adduction. The deformity resembled that of tuberculous hip disease (*Figs. 200, 201*). He was re-admitted



FIGS 200, 201—Case 8, Nov, 1935 The figures show a flexion-adduction deformity with shortening of the limb, what one would expect typically in a chronic tuberculous arthritis



FIG 202—Case 8, Nov 14, 1935 Non tuberculous arthritis of the left hip The pelvis is tilted on account of the deformity shown in Fig 201 The disease has been present for 5½ years There is hardly any destruction of the articular surfaces The joint space has re-formed and some dense new bone formation is to be seen about the lower lip of the acetabulum and the lower articular margin of the femur The joint is quite painless, and mobile under a general anæsthetic.

to the Sanatorium for corrective treatment, but interference with the joint has been discouraged because we feel sure that the condition is not tuberculous and that correction should eventually be obtained by conservative measures. X-ray examination now shows no further destruction of bone, an apparent re-formation of the cartilage space, and some new dense bone formation about the lower lip of the acetabulum and the lower third of the articular surface of the femur (*Fig. 202*). Under a general anaesthetic, the left hip, though flexed to about 45° , was quite definitely mobile, about 15° of flexion, 10° of abduction and adduction, and a little rotation being easily obtained. The Wassermann reaction is negative, but he still reacts to tuberculin, a $1+$ reaction being given to the first test dose of Tuberculin P.P.D. His general condition is very good. There is no pain, and despite the deformity he goes about actively and comfortably.

This patient has some features in common with the preceding *Case 7*, although one joint only was affected. The gradual resolution of the arthritis without bone destruction resulted, in each case, in a stiff though painless joint. Evidence of a quiescent glandular tuberculosis in each case and the onset of symptoms referable to the hip-joint led to the faulty diagnosis of tuberculous hip disease. It is possible that the septic state which produced subcutaneous abscesses was also responsible for the arthritis.

Case 9.—M. P., female, aged 22 years, was a patient in East Fortune Sanatorium from Sept. 4, 1926, to Sept. 21, 1930. She was a tuberculous subject, and had had tuberculous glands removed from the right side of the neck some years previously. Many palpable glands were present in other areas of the neck and in the abdomen. The Mantoux test was strongly positive.

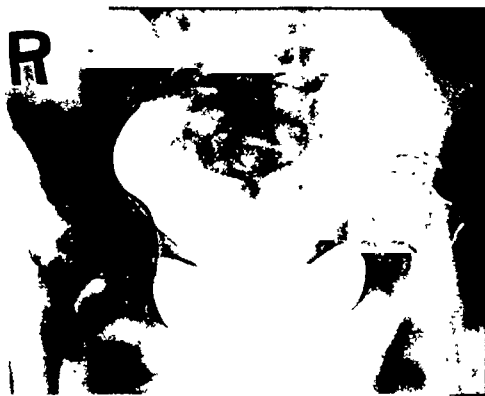


FIG. 203.—*Case 9*, Aug. 29, 1934. Bilateral protrusio acetabuli. The patient had suffered from an arthritis affecting both hip-joints, but the protrusio acetabuli was present to the same degree at the first radiographic examination at the onset of symptoms 8 years previously. There is slight moulding of the head of the left femur, which also shows some cyst-like centres of rarefaction. These features have appeared since the early days of the illness. This patient was tuberculous, but there is nothing to prove that the arthritis was also tuberculous.

On admission the left hip-joint was fixed in a vicious position and showed all the clinical signs of a tuberculous arthritis. The movement of the right hip-joint later became restricted, and there was creaking in both knee-joints. The left hip seemed to be ankylosed. At the first X-ray examination, on Sept. 5, 1926, the head of the left femur was seen to be deeply placed in the acetabulum and there was intra-pelvic protrusion of the acetabulum. A similar degree of protrusio acetabuli was present on the right side, and several small cyst-like areas of rarefaction were seen in the upper margin of the acetabulum on this side, although at this time the symptoms were confined to the left side. The contour of the articular surfaces of both hip-joints were well preserved. The only alteration of radiographic

appearances observed up to the time of her first discharge in 1930 was a tendency to 'moulding' or 'mushroom deformity' of the left femoral head. This never became severe.

After discharge she was seen on several occasions, and, as the right hip was becoming progressively stiffer, she was re-admitted to the Sanatorium on Aug. 8, 1931. At this time she was much crippled. The movements of the right hip were restricted in all directions to less than 50 per cent of normal range. The left hip was fixed in a position of slight adduction, and in a neutral position as regards flexion and rotation. The right knee was somewhat swollen, suggesting synovial hypertrophy, and the left knee-joint was fixed. No definite X-ray changes were present in the knees. She was treated by T.A.B. injections and by massage and passive movement of the joints, and a slight improvement of mobility of the right hip and right knee resulted. She was discharged on Nov. 17, 1934, and when last seen in December, 1935, she was able to get about with the aid of sticks.

Frequent X-ray examinations had been made. These seemed to show a progressive improvement of the condition of the hips. The joint spaces, previously almost obliterated, reappeared, and the articular surfaces became more clearly defined. In the latest radiograph (Fig. 203), recalcification in the right hip has taken place, but some cyst-like areas of rarefaction are still present in the left femoral head. The protrusio acetabuli has not increased on either side during the eight years over which we have observed the patient. There is no further moulding of the femoral heads.

As an additional point of interest, this patient's father is crippled with some form of arthritis.

Protrusio acetabuli, or the 'Otto pelvis', has been fully discussed by Pomeranz,²⁵ Golding,²⁶ and others. Golding recognizes three types of the condition: (1) Due to growth disturbances, often symptomless until the joint later becomes the seat of an arthritis; (2) Due to infective arthritis or osteo-arthritis of rheumatic type; (3) Due to destructive disease of the joint, e.g., pyogenic, syphilitic, or tuberculous arthritis, or to neoplasm. Schaap²⁷ points out that the condition is met with mainly in women, and that it is usually bilateral. He believes that most cases originate in early life, and that the joints may subsequently be affected by an arthritis. It seems that this has been the case in our patient (*Case 9*), for protrusio acetabuli was present on both sides at the first X-ray examination in 1926, even though there were no symptoms on the right side. The protrusion did not advance, and the only radiographic alterations observed over a period of eight years were general and localized rarefaction of bone (very like that observed in adult osteo-arthritis), narrowing of the joint space with subsequent restoration, and a slight 'moulding' or flattening of the left femoral head. The arthritis also affected the knees, but recovery seems to be gradually taking place.

The girl was a tuberculous subject, a fact which caused confusion in the diagnosis. We believe, now, that she was suffering from a non-specific arthritis, and that the original protrusion of the acetabula was neither of tuberculous nor of arthritic origin.

6. Modification of the Course of Non-specific Arthritis due to Early Immobilization

There are, as yet, no certain rules governing the treatment of chronic non-specific arthritis. On the one hand, active exercise must be limited to prevent undue discomfort and exacerbation of the local disease, and on the other hand immobilization, unless under assiduous orthopædic control, is to be avoided as tending to promote early ankylosis and otherwise preventable deformity. We have already tentatively suggested (*Case 1*) that rest and the promotion of general

hygiene, as practised in sanatoria in this country, may delay the dissemination of an arthritis to other joints, but we have also shown that prolonged immobilization is detrimental to non-tuberculous arthritis of the hip. The cases that we shall now describe suggest that immobilization treatment may prevent or delay certain compensatory structural changes which are usually characteristic of the disease. These cases also present some unusual difficulties in diagnosis.

Case 10.—J. S., male, aged 18 years, was admitted to East Fortune Sanatorium on Nov. 8, 1933. In August of that year his left leg had become weak. This weakness was felt suddenly after a walk, and was followed by pain in the groin. In October he was sent to a hospital, where a diagnosis of tuberculous disease of the left hip-joint was made after an X-ray examination. He was then sent to the Sanatorium.

On admission he was in very poor condition, pale, thin, and weak. A presystolic mitral bruit was present. The blood sedimentation rate was increased, and he gave a strongly + reaction to the Mantoux test (1-1000). The blood Wassermann test was negative. The left hip was fixed in flexion at an angle of 45°, adducted and externally rotated, and any attempt at movement caused pain. X rays showed narrowing of the left hip-joint space and slight irregularity of the acetabulum. The right hip-joint was normal, but



FIG. 204.—*Case 10*, Feb. 13, 1936. Arthritis was present in sacro-iliac, hip, and knee joints on both sides. Both hips and sacro-iliac joints are ankylosed, and the picture shows early bony trabeculation across the hip-joints, although the articular contours are well preserved. The case was thought to be one of spondylitis ankylopoietica.



FIG. 205.—*Case 10*, Feb. 13, 1936. Atrophy of the vertebral bodies and almost complete loss of all the intervertebral disc spaces in the lumbar region. Ligamentous calcification was absent—a possible result of prolonged and early immobilization. Shadows of calcareous abdominal glands are to be seen.

the left sacro-iliac joint showed appearances of definite disease. The diagnosis of tuberculosis was accepted and the left limb was placed in extension.

By April, 1934, there were symptoms in the right hip and in both knees. The X-ray film now showed narrowing of the right hip-joint space, and in December, 1934, there was obliteration of both sacro-iliac joints. He had now disease of six joints—sacro-iliac, hip, and knee on both sides. The diagnosis of tuberculosis had been questioned in the early days of his treatment, but it was upheld for a time on account of the strongly positive tuberculin reaction, the presence of calcified glands in the abdomen, and of certain urinary symptoms. A month after his admission the patient had hæmaturia, which was repeated frequently during the following month. In February, 1934, an excretion pyelogram showed dilatation of the pelvis of the left kidney and alteration in the shape of the calyces. The urine contained a little albumin, but no pus and no tubercle bacilli. The hæmaturia was repeated in December, 1934, but did not persist.

He was eventually transferred to an orthopaedic hospital, with no improvement in the physical condition of his joints, which even under a general anaesthetic were fixed. Dr. W. T. Munro investigated his urine and found a few pus cells, but guinea-pig inoculation with the deposit failed to produce tuberculosis. An X-ray film taken on Feb. 13, 1936, showed ankylosis of both sacro-iliac joints and of both hip-joints, with bony trabeculation commencing to show across the hip-joints (*Fig. 204*). The knees showed advanced general rarefaction of bone with 'stencilling' and loss of joint space. The lumbar and thoracic spine, in which there was very little movement, showed atrophy of the vertebral bodies and almost complete loss of all the intervertebral disc spaces (*Fig. 205*). In spite of the absence of calcification of the intervertebral ligaments, the case was now diagnosed as spondylitis ankylopoietica with involvement of the proximal joints of the lower limbs. A diagnostic arthrotomy of the right knee was performed in February, 1936. In this joint the synovial tissues were greatly proliferated and matted together, and there was pannus growth over the articular cartilages. Many histological sections were prepared from tissue removed from the synovial lining of the front of the joint and from the capsule on the inner side of the joint. The general pathological changes were atrophy of the synovial lining, fibrinous degeneration and fibrous-tissue replacement of the synovial tissues, obliterative endarteritis of vessels, and a scattered infiltration of lymphocytes, mononuclear cells, and a few polymorphs. There was no tubercle or giant-cell formation or caseation; neither were there focal collections of lymphocytes. No acid-fast bacilli were found.

Case 11.—A. R., male, aged 26 years, was admitted to East Fortune Sanatorium on June 17, 1933. During 1931 he had pain in the right leg which was called sciatica. After three weeks' rest the pain was relieved, and he resumed work. In December, 1932, he was compelled to give up work on account of pain in the lower part of the back. During the first months of 1933 he was seen as an out-patient at a general hospital. There was some doubt about the diagnosis, the changes shown in the X-ray film being called "infective arthritis or tuberculosis, probably the latter". On this statement, his doctor notified him as sacro-iliac tuberculosis.



FIG. 205.—*Case 11*, June 21, 1933. The sacro-iliac joints are diseased, a condition which was first thought to be tuberculous. There is no radiological abnormality of the hip-joints at this time, although there was limitation of movement of the right hip.

His general condition was fairly good when he came to the Sanatorium. The blood sedimentation rate was raised, and he gave a — reaction to the Mantoux test (1-1000). The Wassermann reaction was negative. He complained of pain in the back, and there was stiffness and rigidity of the lumbar spine. The left hip-joint moved normally. Movements of the right hip-joint were limited to a little passive movement in all directions. X-ray examination showed disease of both sacro-iliac joints, but no abnormality of either hip (*Fig. 206*). The diagnosis of bilateral sacro-iliac tuberculosis was accepted, and he was immobilized in a lumbar plaster. By April, 1934, the right hip-joint was fixed in a position of external rotation. Adduction had been prevented by the plaster. Extension was applied to the right

leg. In December, 1934, movements of the left hip were limited. Later both knees became stiff, and by the middle of 1935 he had exactly the same clinical and radiological condition as that described in *Case 8*—disease of both sacro-iliac joints, both hips, and both knees.

It is most interesting that he also had a similar renal condition. During June, 1934, he complained of pain in the left loin associated with nausea and vomiting. The left kidney was palpable and tender. The urine contained a small quantity of muco-pus, and pus cells and red blood-corpuscles were seen on examination of the deposit. Shortly afterwards he developed scarlet fever, and was transferred to a fever hospital. After his return in July, 1934, he again had loin pain, with similar symptoms, and the urine again contained a little albumin, pus, and red blood-cells. An excretion pyelogram showed pelvic dilatation of the left kidney, and the ureter appeared dilated in its upper course. The kidney was exposed for nephrectomy and was split and explored, but as it appeared quite healthy it was sutured in the usual way and replaced. There were no further urinary symptoms until December, 1935, when hæmaturia recurred, but lasted only a few days. Dr. Munro examined the urine and found a few pus cells in the smears. Guinea-pig inoculation failed to produce tuberculosis.

In March, 1936, the movements of the joints were tested under a general anæsthetic. The right hip was firmly fixed in a position of external rotation. The left hip had slight movement in all directions and adhesions were felt to yield during the manipulation of this joint. The knees had 15° to 20° of flexion movement, but



FIG. 207.—*Case 11*, Feb. 13, 1936. There is now definite evidence of non-ruberculous arthritis of both hip-joints. The right hip was ankylosed and the left hip had only a few degrees of movement when tested under a general anæsthetic. Both sacro-iliac joints are obliterated.

without anæsthesia the patient was rigid from below the knees to the upper thoracic spine. A radiograph on Feb. 13, 1936, showed general loss of bone density and narrowing of the joint space of both hip-joints, and obliteration of the sacro-iliac joints (*Fig. 207*). The spine showed no ligamentous calcification, but irregularities of the lateral margins of the bodies of the 9th, 10th, and 11th thoracic vertebræ suggested early spondylitis (*Fig. 208*).



FIG. 208.—*Case 11*, Feb. 13, 1936. Irregularities of the lateral margins of the bodies of the 9th, 10th, and 11th thoracic vertebræ, together with the ankylosing disease of sacro-iliac, hip, and knee-joints on both sides, suggest spondylitis ankylopoietica. The patient was rigid from below the knees to the upper thoracic spine. No calcification of the vertebral ligaments is to be seen, but this result has possibly been prevented or delayed by the prolonged immobilization.

The correct diagnosis of these two cases (*Cases 10 and 11*) remained obscure for a considerable time. Finally, however, a diagnosis of spondylitis ankylopoietica was established as a result of a study of the literature, and as a result of comparison with another case which recently came to the notice of one of us (D. H. C.). We shall briefly describe this last case, because the symptomatology is rather unusual.

Case 12.—J. J. W., male, at the age of 15 suffered from an attack of arthritis affecting both knees and the right hip-joint. Recovery was apparently complete after a few weeks' rest in bed. At the age of 28 he noticed weakness and disability of the right hip without very much pain. This was not relieved, and in the following year symptoms appeared in the left hip. He received a course of gold injections without benefit, and then was admitted to an infirmary where both legs were placed in extension for five weeks. This treatment was followed by the application of a removable plaster, and massage was given daily for three months. The condition progressed and he was sent to the Harrogate Royal Bath Hospital on Jan. 16, 1936.

On admission there, it was found that both hips and the spine, excluding the cervical region, were quite rigid. The muscles of the thighs, gluteal regions, and back were much wasted. The knees were movable but very stiff. X-ray examination showed an infective type of arthritis of both hip-joints and of both sacro-iliac joints, exactly like the later pictures obtained in *Cases 10* and *11*. The diagnosis, in this case, was simplified by the presence of lateral bridging due to ligamentous calcification, between the 12th thoracic and 1st lumbar, and between the 1st and 2nd lumbar vertebræ (*Fig. 209*). The suspicion of visceral tuberculosis was absent in this case, and he gave no reaction to the first test dose of Tuberculin P.P.D.

Physical and bath treatment enabled him to walk with the aid of two sticks. Pain was relieved, and the adoption of an upright posture restored free movement to his knees. It is doubtful whether progressive ankylosis of the spine and hips can be altogether prevented.

The pathological differentiation of the earlier known cases of spondylitis was made by Knaggs,²⁸ but owing to the scarcity of post-mortem material, which often shows only advanced changes, much of our knowledge of the early stages of this very chronic disease has come from the radiologists. Thus, Scott²⁹ has shown that every one of his 110 cases of 'spondylitis adolescens' had a bilateral affection of the sacro-iliac joints, usually in the form of ankylosis, which may commence some years before the onset of spinal signs or symptoms. Scott is also of opinion that the sacro-iliac infection may cease at any time and never become a spondylitis. Golding³⁰ confirms that sacro-iliac disease always precedes spinal changes, and he has experience of two

instances in which the hip-joints were attacked early with a severe onset and much pain. M. P. Weil³¹ states that the intervertebral discs are often diminished in height, as in our *Case 10*. Forestier³² recognizes that the disease may be active up to five years before the onset of ligamentous calcification. Knaggs²⁸ writes that in the *rhizomélique* form of spondylitis (Pierre-Marie) pain is often located in the lumbar region or in the hips, and that the hips become affected when the disease is active in the lumbar region. Golding mentions the occasional involvement of the knee-joints, and we have observed this in other cases apart from those we have just described.



FIG. 209.—*Case 12*, Jan 27, 1936. Spondylitis ankylopoietica. Calcification of the lateral vertebral ligaments is here shown between D. 12 and L. 1, and between L. 1 and L. 2 vertebræ. The condition of the hips and sacro-iliac joints was identical with that of these joints in *Cases 10* and *11*, but this patient had not been subjected to prolonged immobilization.

Two of our patients (*Cases 10 and 11*) were immobilized for long periods, and although certain abnormalities were revealed radiographically in the vertebral column, neither patient has yet developed ligamentous calcification. Buckley,³³ in an excellent paper on ankylosing spondylitis, makes it clear that calcification of the vertebral ligaments is a defensive reaction designed to counteract the effect of the softening and rarefaction of the vertebral bodies and thus to prevent serious spinal collapse and deformity. The chronic course of the disease allows ample time for this defence reaction to take place. Immobilization treatment adopted in our two cases may well have provided sufficient spinal support to delay the pathological reinforcement of the spinal ligaments. Whether this treatment and its effect are totally beneficial remains to be determined by further experience. In our cases it did not prevent the centrifugal spread of the arthritis from sacro-iliac to hip and knee joints.

The early diagnosis was confused, in *Cases 10 and 11*, by the absence of calcification of the vertebral ligaments, by the early affection of the hips, by the positive tuberculin reactions, and by the occurrence of hæmaturia. We are satisfied that this hæmaturia was not due to genito-urinary tuberculosis, and we can only offer the suggestion that tumefaction around the sacro-iliac joint had caused partial obstruction to the outflow of urine from the left kidney in each case. In *Case 11* the pyelogram revealed dilatation of the upper course of the left ureter as well as of the left renal pelvis.

The question of the ætiology of the spondylitis and arthritis in these cases remains to be considered. Knaggs²⁸ believed a toxic factor to be the cause of ankylosing spondylitis, and it is clear from recent researches that the condition is an infective one of a type similar to rheumatoid arthritis. Gonorrhœa was once considered to be the commonest cause, but many other organisms have now been incriminated. Certain authorities, including Weil³¹ and Forestier³², regard tuberculosis as a possible infective factor in the production of spondylitis, and Buckley, while not subscribing to this view, found that 15 out of 18 of his cases gave a positive tuberculin reaction. Alajouanne and Lacapère³⁴ consider that the evidence for a tuberculous origin is not irrefutable. Be that as it may, ankylosing spondylitis is so clearly a clinical entity and is at such pathological variance with ordinary tuberculous arthritis that its differentiation from the latter in the early stages remains a matter of great importance.

7. The Possibility that there Exists an Atypical Tuberculous form of Polyarthritis—Tuberculous Rheumatism

In 1897, Poncet of Lyons first described the clinical picture of tuberculous rheumatism, and launched a controversy which has persisted, mainly in French medical literature, ever since. In the same year J. Stewart³⁵ of Montreal wrote: "Indirectly a tuberculous tendency may, by lowering the resistance, tend to bring about a rheumatoid arthritis, and it is only in this sense, as I understand it, that there is a connection between the two diseases". Poncet and his school later sought a tuberculous origin for many different forms of arthritis. Around these divergent views the controversy has continued, with little convincing proof on either side, and little attempt at compromise until recent years. Cases described by Bezançon, Bernard, Oumansky, and Gaucher,¹⁷ Laederich, Mamon, Arager

and Léonard,³⁶ Rathery and Doubrow,³⁷ and Le Sage,³⁸ and essays by Ory³⁹ and Weil,⁴⁰ define, more clearly than earlier works, the limitations of the condition which French authors consider to be tuberculous rheumatism.

Under this designation there appear to be two types of disease. The first resembles acute rheumatism, in that the joints are successively affected without permanent lesion or disability, but differs from rheumatic fever in the absence of cardiac involvement and in its resistance to salicylate therapy. This type has been ascribed to a filterable or otherwise altered form of the tubercle bacillus, or to an allergic condition secondary to a visceral tuberculous infection. The second type is that of a transitory or more chronic polyarthritis eventually becoming localized in one of the joints earlier affected. Tubercle bacilli have usually been isolated from the joint in which the infection appeared to localize. In such a case, however, as in our *Case 4*, we cannot be certain that a true tuberculous arthritis has not been superimposed upon a polyarthritis of other ætiology.

Medical authors in this country have paid very little attention to the question. Cases of polyarthritis in tuberculous subjects, which had some points in common with the French cases, have been described by Raw⁴¹ and Copeman and Clay.⁴² For a time, the finding of tubercle bacillæmia by Reitter and Löwenstein⁴³ in many cases of polyarthritis seemed to favour the concept of a tuberculous rheumatism, but negative observations by many other workers, and criticisms⁴⁴ of the technique employed in the original investigations, have belittled the importance of this finding. An excellent and full review of the subject of tuberculous rheumatism has been undertaken by Brav and Hench,²¹ who conclude that there is still no incontrovertible proof of the existence of such an entity as tuberculous rheumatism.

The cases which we have described include a variety of arthritic manifestations occurring in tuberculous subjects. Every investigation and observation that we have been able to make has led us to the conclusion that, with the exception of *Cases 4, 5, and 6*, none of these patients suffered from a true tuberculous arthritis. We cannot categorically deny the possible influence of associated visceral tuberculosis on the production and course of the joint disease, but we prefer not to use the term 'tuberculous rheumatism' for the following reasons: (a) Every case pursued a course consistent with non-specific arthritis; (b) Immobilization treatment, adopted on the suspicion that the condition was tuberculous, proved to be a totally unsuitable procedure in each case.

SUMMARY

The cases of 12 patients suffering from various forms of arthritis have been described. In 11 patients the arthritis was multiple. In 11 patients tuberculosis was suspected, and in 8 visceral tuberculosis was proved. The arthritis in 9 instances was finally not considered to be tuberculous. Six patients were wrongly diagnosed, in the first place, as tuberculous arthritis, and the detrimental effect of treatment adopted on this diagnosis has been recorded. Three early cases of spondylitis ankylopoietica with unusual symptomatology have been included.

The cases have been utilized to illustrate the difficulties in the early diagnosis of tuberculous arthritis and to warn against the too ready acceptance of a tuberculous etiology in multiple arthritis occurring in presumably tuberculous subjects.

The facts which add confusion to the diagnosis of such cases, and which have been discussed and exemplified, are the following :—

1. The insidious monarticular onset of some cases of multiple non-specific arthritis.
2. The coexistence in the patient of some visceral tuberculous lesion, which may or may not influence the course of a non-tuberculous polyarthritis.
3. The possible occurrence of a single tuberculous joint superimposed upon a non-tuberculous multiple arthritis.
4. The occasional incidence of a true tuberculous arthritis in two or more joints.
5. The comparative infrequency of non-specific arthritis of the hip in patients under middle age, and the tendency to suppose such a condition to be tuberculous.
6. Modification of the course of a non-specific arthritis due to early immobilization.
7. The possibility that there exists an atypical tuberculous form of polyarthritis—tuberculous rheumatism.

We believe that confusion in diagnosis in this type of case is not uncommon, and that many other cases of non-specific arthritis may have been subjected to prolonged and expensive immobilization treatment prejudicial to their subsequent recovery of function.

We suggest that in all such cases expectant treatment should be adopted. This should consist of maintenance of general hygiene, and rest in bed without immobilization until unequivocal evidence of the tuberculous or non-tuberculous nature of the joint disease appears, or is obtained by examination of aspiration or biopsy material.

We wish to thank the following physicians of the Harrogate Royal Bath Hospital for access to their patients: Dr. Dorothy Potter (*Case 1*), Dr. Geoffrey Holmes (*Case 4*), Dr. Nimmo Watson (*Case 7*), and Dr. Alfred Sharp (*Case 12*); and we record our gratitude to Dr. W. T. Munro of Glenlomond, Kinross, for his kindness in examining and culturing the urines of *Cases 10* and *11*.

REFERENCES

- ¹ SMITH, A. DE F., and WATERS, W. H., *Jour. Amer. Med. Assoc.*, 1928, xc, 189.
- ² SMITH, A. DE F., *Arch. of Surg.*, 1932, xxv, 54.
- ³ MILGRAM, J. E., *Jour. Amer. Med. Assoc.*, 1931, xcvi, 232.
- ⁴ GHORMLEY, R. K., and BRAY, E. A., *Arch. of Surg.*, 1933, xxvi, 465.
- ⁵ ALLISON, N., and GHORMLEY, R. K., *Diagnosis in Joint Disease*, 1931. London.
- ⁶ GIRDESTONE, G. R., *Brit. Jour. Surg.*, 1931-32, xix, 488.
- ⁷ FORRESTER-BROWN, M., *Ibid.*, 1930-31, xviii, 54.
- ⁸ FRASER, J., *Tuberculosis of the Bones and Joints in Children*, 1914. London.
- ⁹ ROGERS, M. H., *Jour. Bone and Joint Surg.*, 1922, N.S. iv, 679.
- ¹⁰ SMITH, A. DE F., *Jour. Amer. Med. Assoc.*, 1924, lxxxiii, 1569.
- ¹¹ PHEMISTER, D. B., *Ann. of Surg.*, 1924, lxxx, 481.
- ¹² PHEMISTER, D. B., *Jour. Bone and Joint Surg.*, 1925, N.S. vii, 835.
- ¹³ PHEMISTER, D. B., and HATCHER, C. H., *Amer. Jour. Röntgenol.*, 1933, xxix, 736.
- ¹⁴ POMERANZ, M. M., *Ibid.*, 1933, xxix, 753.
- ¹⁵ HENDERSON, M. S., and FORTIN, H. J., *Jour. Bone and Joint Surg.*, 1927, N.S. ix, 700.
- ¹⁶ ATSATT, R. F., *Ibid.*, 657.
- ¹⁷ BEZANÇON, F., BERNARD, E., OUMANSKY, and GAUCHER, *Bull. et Mém. Soc. méd. Hôp. de Paris*, 1931, lv, 742.

- ¹⁸ LONG, E. R., SEIBERT, F. B., and ARONSON, J. D., *Tubercle*, 1935, xvi, 304.
- ¹⁹ COLLINS, D. H., *Jour. Pathol. and Bacteriol.*, 1936, xlii, 113.
- ²⁰ PEMBERTON, R., *Arthritis and Rheumatoid Conditions*, 1935. London.
- ²¹ BRAV, E. A., and HENCH, P. S., *Jour. Bone and Joint Surg.*, 1934, N.S. xvi, 839.
- ²² FORESTIER, J., *Rev. du Rhumatisme*, 1936, iii, 53.
- ²³ FORRESTER-BROWN, M., *Lancet*, 1935, i, 1377.
- ²⁴ TELFORD, E. D., *Ibid.*, 1934, ii, 823.
- ²⁵ POMERANZ, M. M., *Jour. Bone and Joint Surg.*, 1932, N.S. xiv, 663.
- ²⁶ GOLDING, F. C., *Brit. Jour. Surg.*, 1934-35, xxii, 56.
- ²⁷ SCHAAP, C., *Jour. Bone and Joint Surg.*, 1934, N.S. xvi, 811.
- ²⁸ KNAGGS, L., *Brit. Jour. Surg.*, 1924-25, xii, 524.
- ²⁹ SCOTT, S. G., *Brit. Jour. Radiol.*, 1936, ix, 127.
- ³⁰ GOLDING, F. C., *Brit. Jour. Surg.*, 1935-36, xxiii, 484.
- ³¹ WEIL, M. P., *Rev. du Rhumatisme*, 1935, ii, 525.
- ³² FORESTIER, J., *Ibid.*, 472.
- ³³ BUCKLEY, C. W., *Reports on Chronic Rheumatic Diseases*, 1935, i, 77.
- ³⁴ ALAJOUANINE, T., and LACAPÈRE, J., *Rev. du Rhumatisme*, 1935, ii, 390.
- ³⁵ STEWART, J., *Brit. Med. Jour.*, 1897, ii, 1225.
- ³⁶ LAEDERICH, L., MAMON, H., ARAGER, and LÉONARD, *Bull. et Mém. Soc. méd. Hôp. de Paris*, 1931, lv, 1836.
- ³⁷ RATHERY and DOUBROW, *Ibid.*, 1934, lviii, 13.
- ³⁸ LE SAGE, A., *Ibid.*, 284.
- ³⁹ ORY, *Acta Rheumatologica*, 1932, iv (No. xiv), 8.
- ⁴⁰ WEIL, M. P., *Rev. belge Sci. méd.*, 1933, v, 658.
- ⁴¹ RAW, N., *Proc. Roy. Soc. Med.*, 1914, vii (2), (Med. Sect.), 68.
- ⁴² COPEMAN, W. S. C., and CLAY, R. D., *Lancet*, 1935, ii, 1460.
- ⁴³ REITTER, C., and LÖWENSTEIN, E., *Wien. klin. Woch.*, 1934, xlvii, 1569.
- ⁴⁴ SIEGEL, M., *Amer. Jour. Med. Sci.*, 1935, cxc, 345.

GRAVITY DRAINAGE OF PELVIC ABSCESS

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THIS paper has for its object the determination of *precisely* what place the rectal or vaginal drainage of abscesses in the pouch of Douglas should occupy in surgery. It seems fair to say that to the average practitioner this operation presents itself as a simple, safe, and eminently satisfactory procedure. That this is emphatically not the case it is the purpose of this communication to show.

THE LITERATURE

MacLaren¹ (1908) gave the opening paper in a symposium dealing with the question of purulent collections in the pelvis. He stated that the problem had been solved in the female, in whom such cases could be cured by section per vaginam. He drew attention to the great importance of catheterizing the patient before opening an abscess through the rectum, and mentioned a case where he opened the bladder in error through omitting this precaution. Although the abscess was opened also, the patient recovered. He was the first to give the warning that dangerous bleeding might occur through division of a branch of the superior hæmorrhoidal artery in the rectal wall. He advised that in all cases of diffuse septic peritonitis, after the third day, whether the rectal wall was bulging or not, an incision should be made into it, a finger inserted, and the pelvis explored for pus. He terminated his address by saying: "And what is the danger of rectal drainage? None at all according to a limited experience." These remarks were generally agreed to in the ensuing discussion.

Ritchie² (1918) states that any uncertainty of diagnosis whether there exists an abscess into the pouch of Douglas should lead to postponement of the operation of rectal drainage or the use of other measures. The operation should be strictly limited to cases with a well-defined mass per rectum. Here also the advice is given to drain away the exudate in general peritonitis by rectal incision. Obviously at the dates quoted the pathology of the peritoneal exudate was not appreciated.

Ritchie stated, moreover, that the operation should be confined to males and children, as it presents no advantage over section per vaginam. He says emphatically that the dangers and risks of rectal drainage are not to be considered, because of the usually imperative circumstances. He drew attention to the important fact that because of the adjacent inflammation the patient could not completely empty the bladder, making pre-operative catheterization more imperative than ever.

Moynihan³ (1918) wrote that an abscess which bursts into the rectum or vagina drains very freely and closes more rapidly than if drained from above. He used the rectal route on three occasions in conjunction with a suprapubic drain, and stated that healing was much more rapid than it would have been if only one incision had been used.

Carson⁴ (1924) stated that discharge ceases quickly after rectal drainage has been instituted, and that it is characteristic of these cases that recovery is rapid. He also stated that the small intestine is pushed up by the collection of fluid and that there is little risk of its being injured. He said: "I have never known any accident happen as a result of the rectal incision."

Blake⁵ (1928) expresses a sharply contrary opinion: "Pelvic abscesses in the male are best drained, as a rule, by suprapubic incision; exceptionally they may be drained by *perineal incision*, the dissection being carried up between the rectum and the prostate. These abscesses can be drained into the rectum, but this procedure is objectionable; therefore it is often unsatisfactory and the dressings are painful.

Hamilton Bailey⁶ (1930) says that twenty or thirty years ago drainage of pus per rectum was extensively done, but the practice has fallen into disfavour and has now been almost entirely supplanted by suprapubic drainage. The reasons for the change which he gives are, firstly, that a loop of obstructed small gut in the retro-vesical pouch may be mistaken for a collection of pus, and, secondly, that a pelvic collection of pus does not necessarily mean that this collection is shut off by adhesions in the pelvis. He stigmatizes the procedure as a blind one which has justly earned the reputation of being dangerous. Blind rectal drainage should therefore be reserved for those cases where the history, the free discharge of mucus, the soft cystic swelling, and complete absence of rigidity, make the diagnosis of a localized abscess pointing in the rectum undeniable. If the slightest doubt exists, laparotomy should be done. Should this disclose an abscess localized in the pelvis, he advises closure of the suprapubic wound and rectal drainage.

Kirschner⁷ (1933) says that abscesses in the pouch of Douglas are preferably opened through the rectum or vagina.

Burnett⁸ (1934) states that this procedure fell into disrepute because it is a blind procedure and cases were not carefully chosen. There are times when this procedure may save the patient, who would die from the toxæmia of an infection too slowly relieved by uphill suprapubic drainage. He points out that abdominal drainage is a major procedure, with its risks of anæsthesia, shock, and the spread of infection. Rectal or vaginal drainage is a brief minor procedure in proper cases and avoids most dangers. He says that the rectal wound tends to close in two or three days and the finger must be inserted daily to avoid this.

CASE REPORTS

Of 10 cases of pelvic abscess drained per rectum or vaginam, 7 cleared up rapidly and uneventfully. Three cases were followed by serious complications. These three patients died. In two of these cases, however, complications following drainage of the abscess were not the only causes contributory to the final issue.

Case 1.—L. K., married, age 34. Patient was a small frail woman who complained of lower abdominal pain and ill health for some weeks. Ultimately a P.V. examination disclosed a cystic swelling in the pouch of Douglas. At this time the temperature was 102°, the pulse 110, and the white blood-count was 15,000 with 85 per cent polymorphs.

Under general anæsthesia a needle was passed into the bulging posterior fornix and thick creamy pus aspirated. A knife was passed into the swelling in the mid-line. Much creamy fluid escaped mixed with hair, and lastly a coil of small bowel prolapsed into the vagina. The opening into the vaginal vault was sutured after the gut had been returned into the peritoneal

cavity, and abdominal section was performed at once. The collapsed dermoid cyst was removed, a pelvic appendix lying in a cavity of encysted pus was removed, and the abdomen was drained by the suprapubic route. The patient slowly developed the rare condition of extra-peritoneal cellulitis, which resulted in pockets of pus forming in the extra-peritoneal fat till ultimately the outer surface of the peritoneum was surrounded with pus cavities. After a lingering illness of six months' duration the patient died.

Comment.—In this case the diagnosis was at fault. All the signs indicated abscess formation; the cystic swelling filled the pelvis, the exploring needle showed pus-like fluid. Lowered resistance in a frail unhealthy woman supplied the other factors necessary for the extensive spread of the infection, once the integrity of the peritoneum was disturbed by the opening of the parametric cellular tissue when the cyst was removed.

Case 2.—M. L., aged 27. Patient was admitted to the Johannesburg General Hospital under my care suffering from acute obstructive appendicitis. The appendix was removed intact. Drainage was not used. On the eighth post-operative day the temperature, which had been normal for six days, began to rise. The lower abdomen became tumid. Mucus was present in the stools, a pelvic collection could be felt per rectum. The patient's general condition was low. It was resolved to wait until an abscess bulged the rectal wall, which state of affairs obtained three days later.

Operation was performed with the patient in the lithotomy position, the bladder having been emptied by catheter. The anterior rectal wall tensely bulged into the lumen of the bowel. It was punctured with a Christopher Martin trocar, the opening being enlarged by inserting one finger. About two pints of foul-smelling pus were evacuated. The finger was put into the rectum to feel if everything was satisfactory, when a soft structure was detected. Examination with a speculum showed this to be a *knuckle of small bowel*. It was quite impossible to rectify this through the anus. An immediate abdominal section had to be done in a patient already much reduced by suppuration in the abdomen.

Operation was rendered notable by two unusual features:—

1. It was only with the greatest difficulty that the pelvic abscess cavity could be found, as the gut was matted in a dense roof extending across the pelvic brim.

2. Between this roof and the right side of the pelvic brim existed a fissure through which a loop of bowel extended, this loop lying on the pelvic floor, i.e., forming part of the contents of the abscess cavity. It was this loop which prolapsed into the rectum. Filmy adhesions prevented pus from the abscess escaping into the general peritoneal cavity alongside the loop of bowel which was prolapsed through the fissure which has been described. Because of the matting together of the intestines, the technical difficulties, and the seriously depleted general health of the patient, it was considered advisable to cover the hole in the rectum with gauze packing, several yards being necessary. The end of the plugging was brought through a suprapubic stab for drainage purposes. This plugging was slowly removed between the sixth and eighth post-operative days. There was no further protrusion of the bowel into the rectum, but a fæcal fistula had developed. The patient was unable to overcome the infection, dying from weakness due to continued suppuration a month after operation. Knowing the matted state of the abdominal contents it was considered inadvisable to do a colostomy. In this case rectal drainage of a pelvic abscess ultimately caused the death of the patient.

Comment.—This case is noteworthy. No record of a similar case can be found in South African medical libraries. There seems no reason to doubt that a loop of small gut lay in the pelvis *in an abscess cavity bathed in pus*, yet functioned normally. There was nothing whatsoever to indicate this state of affairs before operation. The only way it could have been demonstrated would have been by radiography and following the barium through the bowel. Furthermore, it seems that if and when pelvic abscesses are drained per rectum or vaginam a complete barium examination should precede the drainage. If bowel (excluding the rectum) is seen in the pelvis, rectal or vaginal drainage is contra-indicated.

Case 3.—C. F. B., surgeon. This patient was operated on by Mr. Dauth for recurrent attacks of colic, an upper abdominal mass being present. A carcinoma of the transverse colon was disclosed. Carcinoma cells had carried infection through the bowel wall, an abscess being enclosed by omentum, transverse mesocolon, and small bowel. The affected loop of colon was delivered and the wound closed. The exposed bowel was removed with the cautery next day.

The patient slowly went downhill—the wound suppurated and gaped, and a jejunal fistula formed. He developed a large pelvic abscess, and there was three-quarters of an inch of tissue between the lumen of the rectum and the abscess cavity; a tube was fixed in the cavity by a stitch at the anal margin. This caused excruciating pain and ultimately a perianal abscess. The patient, a brilliant surgeon, stated during his illness that silkworm gut should never be used in infected tissues because it was too rigid and caused too much pain, and that the tube draining a pelvic abscess should never be fixed with a stitch.

It was found necessary to reopen the drainage track with the finger on several occasions as it tended to close. Within two days of the initial drainage of the abscess an intensely painful acute cystitis developed. This caused the most excruciating suffering, due to the contraction of the inflamed thickened bladder wall. The patient died after a protracted illness, during which fearful agonies were borne with unexampled fortitude.

Comment.—In this case it was entirely impossible to utilize any other method of drainage than per rectum. The state of the abdominal wall and the precarious condition of the patient alike precluded abdominal section. The noteworthy features of the pelvic abscess were the thick wall which constantly closed the drainage hole, and the spread of the infection through the bladder wall, causing cystitis. The peritoneum acts as an able localizing agent, but once the peri-vesical connective tissue became infected where it was exposed in the drainage track, the infection was able to reach the bladder and spread through its wall and cause cystitis. This spread was by direct extension. Frequency of micturition, dysuria, and diarrhoea are classical symptoms of pelvic abscess, being due to the irritation of bowel and rectal walls by hyperæmia as they form part of the periphery of the abscess. It is surprising how rare a frank cystitis is in cases of pelvic abscess.

In this case the rectal drainage of the abscess led to a painful complication but was not responsible for the patient's death.

DISCUSSION

No mention so far has been made of the cases of pelvic abscess which do well after rectal or vaginal drainage. No object will be served by giving case histories, but there is one factor of supreme importance which was common to all cases, the ages varying from 4 to 70 years, this factor being that none of these patients were gravely ill. True, all had had a serious illness—acute appendicitis with localized peritonitis; but all had rapidly got better of the infection and looked and felt well despite the existence of an abscess. It seems correct, then, to say that a patient who is gravely ill is suitable soil for the implantation of some serious or fatal complication following the making of a drainage opening anywhere, whether this be suprapubic, vaginal, or rectal.

The most serious complication which may ensue on pelvic drainage is presented by the small bowel. Had the loop of bowel lying on the pelvic floor in *Case 2* been adherent to the anterior surface of the rectum it might quite well have been opened by the trocar. The lesson to be learnt from the case is that only by X rays can the presence of such a dependent loop be demonstrated. Though the complication is rare, it is serious, as the prolapse of the small bowel into the rectum will almost

certainly lead to a fatal issue. It is of the first moment to observe that abdominal section may fail to disclose the fact that a loop of small bowel is lying on the floor of the pelvis (*Case 2*). This measure, therefore, is not an absolute safeguard against injury to the bowel during the process of instituting gravity drainage. It follows that cases of pelvic abscess suitable for rectal drainage should have careful X-ray examination first wherever this is possible. If the patient is too ill, as sometimes happens, then he is certainly too ill for any but rectal drainage, and the attendant risks must be taken.

As the result of the foregoing analysis, the following facts emerge :—

1. Rectal or vaginal drainage of a pelvic abscess is a valuable therapeutic measure.
2. In by far the greater number of cases the operation is followed by no ill effects and recovery is prompt.
3. In persons already gravely ill the procedure may be followed by cystitis, extra-peritoneal cellulitis, etc. Yet this is the very type of case where the indications for gravity drainage are the least contentious.
4. The two main risks are : (a) Mistakes due to faulty diagnosis ; (b) Injury to the small bowel.
5. If the diagnosis is the least in doubt, then abdominal section is necessary, followed if indicated by gravity drainage per rectum or vaginam.
6. Extremely rarely the small bowel may herniate through the drainage opening. Should it prolapse into the vagina there is not much harm done providing the emergency is promptly and suitably dealt with. Should the prolapse occur into the rectum, the issue is likely to be fatal.

There are certain negative rules :—

1. *Never* drain per rectum or per vaginam if the diagnosis is in doubt, without preliminary abdominal section.
2. *Never* perform the operation unless the catheter has been passed on the table.
3. *Never* drain through any but an opening exactly median in position.
4. *Never* stitch the drainage material to the anus.
5. *Never* drain below if the abscess in the pelvic cul-de-sac bulges into rectum or vagina on one side only. The abscess which admits of gravity drainage must bulge the rectum or posterior fornix in the midline, i.e., it must be large enough to fill the pouch of Douglas completely.

If the occasional execution of an innocent person is no argument for the abolition of capital punishment, then, surely the very occasional occurrence of a serious complication following gravity drainage of a pelvic abscess should not be permitted to condemn a procedure which is usually simple, expedient, and eminently satisfactory.

REFERENCES

- ¹ MACLAREN, A., *Surg. Gynecol. and Obst.*, 1908, vi, 671.
- ² RITCHIE, H. P., *Jour. Amer. Med. Assoc.*, 1918, Aug. 10.
- ³ MOYNIHAN, SIR B., *Abdominal Operations*, 1918, ii, 179. London.
- ⁴ CARSON, H. W., *Modern Operative Surgery*, 1924, ii, 28. London.
- ⁵ BLAKE, J., *Nelson's Loose-leaf Surgery*, v, 32.
- ⁶ BAILEY, HAMILTON, *Emergency Surgery*, 1930, ii, 28. Bristol : John Wright & Sons.
- ⁷ KIRSCHNER, MARTIN, *Operative Surgery (The Abdomen and Rectum)*, 1933, 341.
- ⁸ BURNETT, W. E., *Surg. Clin. N. Amer.*, 1934, xiv, No. 1, 155.

MYOMATA OF THE ŒSOPHAGUS

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SIMPLE tumours of the Œsophagus—lipoma, fibroma, adenoma, papilloma, and the various cystic epithelial tumours—are all rare. These tumours are as a rule small, they do not cause symptoms, and are usually chance findings at autopsy. The clinical insignificance of these growths, and the lack of features of special pathological interest, are no doubt factors in the comparative infrequency with which such tumours are recorded. The following case of multiple myomata of the Œsophagus is deemed worthy of report as the clinical symptoms were marked and the tumours were the primary cause of the death of the patient.

CASE REPORT

A man aged 74 years was admitted to the Royal Victoria Infirmary, Newcastle-upon-Tyne, as an emergency suffering from severe dysphagia and asthenia. He was in such a weak condition that no satisfactory history could be obtained, and a palliative gastrostomy was performed by Mr. Andrew Logan after a diagnosis of carcinoma of the Œsophagus had been made. His general condition showed progressive deterioration, and death occurred three and a half days later. Owing to his physical state, no radiological or laboratory investigations were made. Attempts to obtain a proper clinical history from the patient's medical attendant failed, as the latter did not reply to the inquiry.

POST-MORTEM EXAMINATION.—This was performed six hours after death. The body was extremely wasted and dehydrated. The following is a summary of the findings:—

Stomach.—Fresh easily broken-down adhesions present between the stomach and the anterior abdominal wall in the region of the left costal margin. The organ was of normal size, with some slight thickening of the mucosa, and early post-mortem digestion over the fundus. On the lesser curve, some 6 cm. from the pylorus, were three healed linear scars. The pylorus was slightly narrowed by a small rounded nodular tumour in the posterior wall which, on section, showed a fasciculated structure suggestive of myoma or adenomyoma. Anterior gastrostomy opening healthy, no leakage.

Pleural Cavities.—Numerous dense old adhesions on both sides, especially at the right base; cavities practically dry.

Trachea.—Full of a brownish froth; mucosa roughened and injected.

Left Lung.—Showed small areas of broncho-pneumonic consolidation throughout. The apex was a mass of scar tissue, and scattered in this were several pea-sized active phthisical cavities.

Right Lung.—Showed small areas of broncho-pneumonic consolidation as in the left, with congestion and a little œdema. The upper lobe was hard and solid and studded with small rounded greyish nodules of active tuberculosis.

Pericardium.—Healthy, no excess of fluid.

Heart.—Normal size, dilated right side, aortic and mitral valves show some thickening with a little fibrosis of the tips of the papillary muscles. Myocardium a little softened; coronary vessels healthy; the first part of the aorta had a slight degree of atheroma.

Liver.—No abnormal features apart from some toxic swelling and pallor.

Gall-bladder.—Contained about 60 to 70 mixed faceted stones of various sizes.

Suprarenals.—No obvious pathological change.

Kidneys.—Not reduced in size; slight senile atrophy, but no signs of renal lesions; pelvis slightly dilated, no inflammation.

Bladder.—Muscular wall was hypertrophied and showed many trabeculæ; mucosa a little hyperæmic; ureters—slight generalized dilatation.

Prostate.—Slightly enlarged throughout, very nodular; on section, white and meaty, suggestive of carcinoma.

Spleen.—Rather small; sugar-icing thickening of capsule; adherent to diaphragm; somewhat atrophic on section, with prominent fibrous trabeculæ.

Œsophagus.—Externally the œsophagus was not unduly adherent to the mediastinal structures, but the lower end was thickened, and in the posterior wall of the middle third several small discrete hard rounded swellings were felt. It was laid open along its posterior

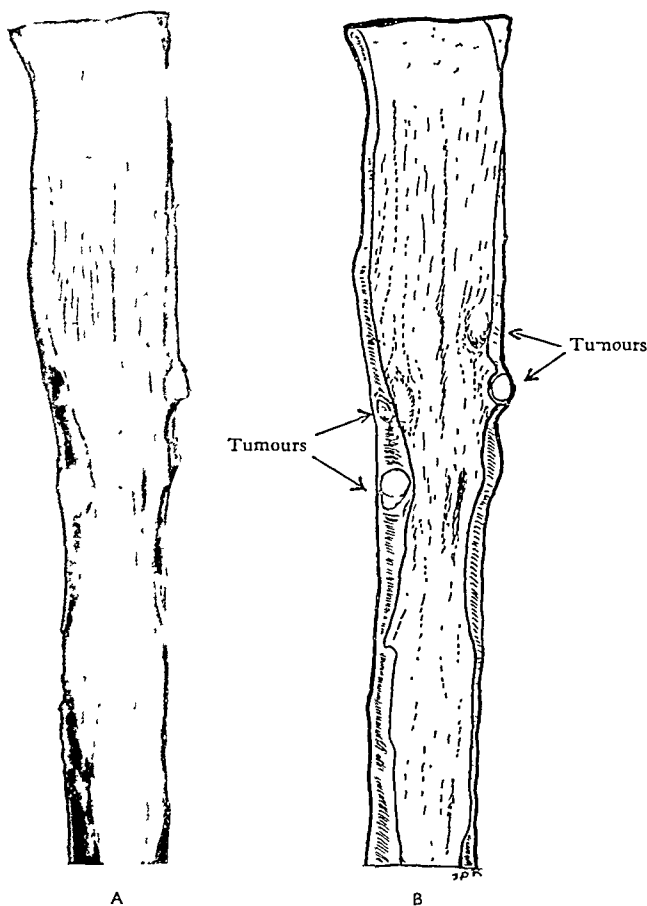


FIG. 210, A, B—Œsophagus laid open posteriorly. Two of the tumours are seen interrupting the continuity of the circular muscle coat. The dilatation above the growths is indicated by the linear folds of the mucosa. The hypertrophy of the circular muscle below the tumours is clearly seen. The distal 3 cm. of the œsophagus is not shown.

aspect, dissected up from the trachea and pericardium, and pinned out for fixation in 10 per cent formalin saline (Fig. 210, A, B). The fixed specimen, including the distal 2 cm. which had been removed with the stomach, was 30 cm. in length. Four small, nearly spherical nodules were situated at the junction of the upper and middle thirds, the most proximal being 9 cm. from the cricoid, the remainder within the next 3 cm. Of the four nodules, three were spherical, with diameters of 0.5, 0.7, and 0.8 cm.; the other was slightly elongated in the length of the œsophagus, and measured 0.8 × 0.6 cm. The tumours were hard, of almost cartilaginous consistency, and the cut surfaces were greyish-white in colour with a watered

silk appearance, sharply contrasting with the brownish muscular coats. They were all situated in, and interrupted the continuity of, the circular muscle. The tumours were not adherent to the mucosa or submucosa, which were freely movable, and the mucosa appeared healthy throughout, apart from a slight hyperæmia in the immediate vicinity of the growths. The œsophagus at the upper end had a normal circumference of 4 cm. At the level of the first nodule it had decreased to 3 cm., this being further reduced to 2.7 cm. at the most distal nodule. It was noted that the mucosa between the cricoid and the first tumour was redundant and thrown into many more longitudinal folds than are usually found, indicating that during life there had probably been here some degree of dilatation. Just beyond the lowest nodule the diameter of the œsophagus narrowed down to 2 cm., after which it again increased to 2.7 cm. for a further 4 cm. before narrowing to 1.8 cm. just above the cardiac opening. At the upper end the circular muscle coat measured 0.2 cm. in thickness, while at the cardiac end it was 0.7 cm. The longitudinal muscle was of a uniform thickness of 0.15 cm. throughout.

Microscopical examination of the tumours (Figs. 211-214) showed that they had the structure of pure myomata, being composed of bundles of smooth muscle fibres running in all



FIG. 211.—The junction between the tumour and the circular muscle. Some of the muscle fibres can be seen merging into the growth. (*Low power.*)

directions, with a minimum of connective stroma. Blood-vessels were extremely scanty and had very thin walls. The muscle fibres were similar to those of ordinary unstriated muscle, but were distinctly longer and thicker. The nuclei were uniform in size and shape, but were plumper than the normal round-ended rod-shaped nuclei of plain muscle, and apart from this increase in size of the nucleus and cytoplasm they were indistinguishable from those of the adjoining circular muscle fibres. The cellularity of the tumour appeared rather less, however, because of this increase in thickness of the individual fibres. No large areas of degeneration or cellular infiltration were present, but many of the tumour fibres showed a peculiar nodal swelling of the cytoplasm. About half the length of the cell was swollen to twice the usual diameter, and this stained more deeply with eosin. Cells showing this change were present but scantily in the section, but they tended to appear in adjacent fibres, producing a palisade appearance. The remainder of the cytoplasm of the affected muscle fibres seemed proportionately thinner, giving the impression that part of the swelling was due to a focal condensation of the cytoplasm. The change would appear to be a degenerative one, as the nuclei of the affected fibres were swollen and stained more deeply. Similar but less marked



FIG. 212.—The margin of the tumour. Here it is clearly demarcated from the circular muscle. (*Medium power.*)



FIG. 213.—The centre of the tumour. Showing the typical whorled structure of the myoma, with bundles of smooth muscle running in different directions. (*Medium power.*)



FIG. 214.—Showing the peculiar oval swellings of the cytoplasm of some of the muscle fibres. (*High power.*)

changes were found occasionally in the neighbouring circular muscle coat. The demarcation of the tumours from the circular muscle was generally good; over the greater part of their circumference a thin line of collagenous fibres was to be found, but at several points this could not be recognized, as the outermost fibres of the tumours—which mainly ran circularly—appeared to mingle and fuse with those similarly arranged fibres of the circular muscle.

Microscopic examination of a transverse section of the lower end of the œsophagus showed the epithelium of this section, which was taken about 4 cm. from the cardiac orifice, of the usual squamous, non-keratinizing type; it appeared to be slightly thicker than usual, but presented no pathological features of note. The lamina propria was seen to be of normal thickness, and consisted of rather dense irregularly arranged bundles of collagen which were rather coarse. The capillary blood-vessels were numerous, and some showed a perivascular round-celled infiltration. The muscularis mucosæ was definitely increased in thickness, but otherwise showed no special changes. In the slightly thickened submucosa the collagen fibres were more loosely arranged, and no signs of inflammation were seen. The circular muscle was from three to four times the normal thickness, and both the number and width of the muscle bundles were increased. Some of the fibres showed the bullous hyaline swellings which were noted in the tumours as giving the appearance of a nodal arrangement of the cytoplasm with pale staining areas between them. In longitudinal section similar palisading was noted. Auerbach's plexus was noted to be much more prominent than usual. The longitudinal muscle was hypertrophied, about two-thirds the thickness of the circular muscle, and showed the same nodal degenerative features. The outer fibrous tissue coat exhibited no special pathological change.

SUMMARY OF REPORTED CASES

Table I.—REPORTED CASES OF MYOMA OF THE ŒSOPHAGUS

AUTHOR AND DATE	SEX AND AGE	NUMBER	SIZE AND SITE	SYMPTOMS	REMARKS
Monro 1797	—	2	—	—	Constricted the œsophagus
Charles Bell 1816	—	2	(a) At level of cricoid (b) Middle of œsophagus	—	—
Lippich 1838	—	1	2 × $\frac{1}{2}$ in., 4 in. proximal to the cardia	—	Ulcerated into trachea at level of bifurcation
Virchow 1863	—	1	12 mm., in lower third	—	Arose from muscularis mucosæ
Eberth 1868	F. 50	1	9.1 × 11.9 / 3.5 cm., in posterior wall, embracing lower third	—	Horseshoe-shaped and embraced œsophagus
Coats 1872	M. 61	1	2 × 4 $\frac{1}{2}$ / 1 $\frac{1}{4}$ in., posterior surface 7 in. from glottis	Dysphagia	Direct cause of death. Pedunculated
Rolleston 1875	F. 49	1	Cherry-sized, junction of upper and middle thirds	—	Originated in circular muscle
Tonoli 1880	M. 70	1	Hazel-nut-sized, middle third	Dysphagia and vomiting	Pedunculated and caused obstruction to food, and œsophageal dilatation present above the tumour
Trastour 1881	M. 56	1	Small egg, inferior part	—	Calcified, and compressed descending aorta

Table I.—REPORTED CASES OF MYOMA OF THE ŒSOPHAGUS—*continued*

AUTHOR AND DATE	SEX AND AGE	NUMBER	SIZE AND SITE	SYMPTOMS	REMARKS
J. Meyer 1882	F. 78	1	8 cm., posterior wall of upper third	—	Dilatation and hypertrophy of œsophagus above the tumour. Displacement of mediastinal structures
Reher 1885	F. 43	1	Hazel-nut-sized, inferior part	—	Pedunculated
Phillip Meyer 1887	—	1	2 × 7 cm., anterior wall 3 cm. below tracheal bifurcation	—	—
Illig 1894	—	1	5 × 5 × 2 cm., posterior wall at level of bifurcation of trachea	—	Calcified, originated from internal muscle layer
Pichler 1897	M. 50	14	Two in middle part, rest about cardia	—	Connected with the circular muscle
Fagge 1899	M. 38	1	2 × 1½ × 1 in., middle third	—	Originated in longitudinal muscle
Maucher (1) 1900	—	1	3 × 1.5 × 2 cm., just below tracheal bifurcation	—	—
(2)	—	1	2 × 5 cm., anterior wall	—	—
Kraus 1902	—	1	—	—	—
Bryant (1) 1905	M. 60	1	Chestnut-sized, cardia	—	Originated in outer coat
(2)	F. 62	2	(a) 3.2 × 3 × 2.5 cm., 14 cm. below pharynx (b) 1.4 × 0.7 × 0.5 cm., at cardiac end	— —	Narrowed lumen of œsophagus Opposite to this was an ulcer
Orth 1909	M. 27	1	Surrounded cardia	—	Ring-like structure
Frank (1) 1911	M. 46	1	Plum-sized, anterior wall at level of bifurcation	—	Bi-lobed, mucosa covering it was exfoliated, exhibited perivascular round-celled infiltration
(2)	M. 60	2	(a) Cherry-sized, just above bifurcation, anterior wall (b) Little smaller and at bifurcation	— —	Circumscribed areas of calcification —
Anitschkow(1) 1911	—	1	Hazel-nut-sized, cardiac end	—	Submucous site
(2)	—	1	Hen's-egg-sized, anteriorly and laterally about cardia	—	Originated in outer muscle layer
(3)	—	1	Bean-sized, laterally at level of tracheal bifurcation	Symptoms of stenosis	Diagnosed as a carcinoma

Table I.—REPORTED CASES OF MYOMA OF THE ŒSOPHAGUS—continued

AUTHOR AND DATE	SEX AND AGE	NUMBER	SIZE AND SITE	SYMPTOMS	REMARKS
Gajet 1911	F. 75	1	Almond-sized, above cardia	Dysphagia	Pedunculated, directly responsible for death
Milanovic 1914	F. 76	1	Nut-sized, anterior wall at level of tracheal bifurcation	—	Calcified, projected into lumen
(2)	M. 25	1	10 × 6 cm., anterior wall 4 cm. from cardia	—	Associated with a diverticulum of œsophagus
(3)	M. 68	2	(a) Hazel-nut-sized, right posterior wall 3.5 cm. from cardia (b) 6 × 5 cm., just above, in posterior wall	—	Associated with a diverticulum
(4)	M. 63	1	Hazel-nut-sized, at cardia	—	Associated with a gastric myoma
(5)	M. 42	1	Hen's-egg-sized, anterior wall at level of tracheal bifurcation	—	Lobulated by a fissure
Hall 1916	F. 17	1	Diffuse through nearly whole œsophagus	Dysphagia	Probably a sarcoma
Bauer 1917	(1) M. 41	1	Plum-sized, posterior wall of upper third	—	Projected into lumen, vascular and œdematous
(2)	M. 38	1	Pea-sized, posterior wall	—	—
Tschlenow 1922	(1) F. 20	Multiple nodules	23 × 9 cm., 3 cm. from cricoid	—	Associated with gastric myoma
(2)	M. 25	2	(a) 1.5 × 1 × 1 cm. (b) 2.5 × 2.5 × 1 cm. Both (a) and (b) at level of tracheal bifurcation	—	Tumours united by a bridge of tumour tissue
(3)	F. 71	1	4 cm. in diameter, just above cardia, with a tail 3 × 1 × 1.5 cm. around cardia	—	Arising from circular muscle, and tends to surround the œsophagus
(4)	F. 62	1	1 × 0.5 mm. at tracheal bifurcation	—	—
Stewart 1931	(1) F. 32	1	3.5 × 1.5 × 1.3 cm., 2.5 cm. above cardia	—	Associated with a diverticulum from circular muscle, some local hypertrophy
(2)	F. 59	1	1.5 × 1 × 1.5 cm., 1.2 cm. above cardia	—	Associated with a diverticulum and from circular muscle
Spitznagel 1931	(1) F. 64	1	6 cm. long, in lower part	—	Calcified tumour
(2)	M. 68	1	Child's-fist-sized with a peduncle, 7 × 8 × 5 cm. and pedicle 2.5 cm.	Dysphagia	Long pedicle, diagnosed substernal goitre
Bezza 1932	(1) M. 68	1	Hazel-nut-sized, 6 cm. from cardia	—	Arising from circular muscle

Continued on next page

Table I.—REPORTED CASES OF MYOMA OF THE ŒSOPHAGUS—*continued*

AUTHOR AND DATE	SEX AND AGE	NUMBER	SIZE AND SITE	SYMPTOMS	REMARKS
Bezza 1932	(2) M. 63	2	(a) Cherry-sized, at tracheal bifurcation, posterior wall (b) Slightly larger, 3 cm. from cardia	—	Projected into lumen
	(3) M. 73	1	Pea-sized, in posterior wall a little above cardia	—	Arose independent of muscle coat Slight muscular hypertrophy above and below. Arose from longitudinal muscle
	(4) M. 35	1	Rice-grain-sized, in posterior wall in upper third	—	Arising from muscularis mucosæ
	(5) F. 60	1	8.5 × 5 cm., at level of arch of aorta	—	Deviation of lumen of œsophagus
In Museum at Cairo (G. G. Turner)	M. 60	1	Cherry-sized, lower end	—	—

DISCUSSION

A thorough search of the literature has produced 49 cases only of myomata of the œsophagus, the more important features of which are tabulated. In 12 the age was not given, but from the remainder it is evident that the tumours are found more commonly in the aged, 13 of these being between 60 and 70 years. It should be noted, however, that this decade has the highest death-rate from all causes, and as the tumours are as a rule benign and do not cause death, it is to be expected that the incidence would be greatest in this age period.

Table II.—AGE INCIDENCE.

AGE IN YEARS*			NUMBER
10-20	1
20-30	4
30-40	4
40-50	5
50-60	4
60-70	13
70-80	6
			37

* 12 not recorded.

These tumours have been found with greater frequency in the male (22) than in the female (15); in this they resemble the other myomata of the intestinal tract. It has been asserted (Bezza) that this may be due to the greater liability of the male tract to irritation from alcohol and coarse food. But it is very doubtful if there is a real sexual difference in the incidence. All the reported tumours were

found at post-mortem examinations, and in most hospitals the autopsies on males are more frequent than on females. The proportion over the last ten years at the Royal Victoria Infirmary, Newcastle-upon-Tyne, is 1·6 to 1. Bezza, in 1932, based his analysis on 26 cases, and found a distinct preponderance of males over females, 3 to 1, but the addition of cases which he omitted and those reported since that date shows a more equable distribution.

The tumours occurred singly in 35 cases and were multiple in 11.

Unlike carcinoma of the Œsophagus, the tumours may occur at any part of the Œsophagus, but the distal third is more frequently affected. In this series 8 were in the upper third, 18 in the middle, and 28 in the lower third. The posterior wall is a more common site than the anterior, and the left and right lateral walls are the least likely to be affected.

The size of these myomata may vary from a millet seed (Bauer's second case) to a diffuse mass several inches long (Tschlenow's first case, A. J. Hall's case). The largest tumours may become pedunculated, and not unnaturally these are the most likely to give rise to symptoms (4 out of 5). The larger non-pedunculated tumours show a tendency to encircle the wall of the Œsophagus (Anitschkow's second and third cases, Pichler's case, and Bezza's fifth case).

Symptoms referable to the Œsophagus were found in 6 cases only, and 4 of these were pedunculated; the other 2 were both of considerable size and showed a tendency to encircle the Œsophagus (Anitschkow's third case and Hall's case), but there was some doubt as to the benignity of the tumour reported by A. J. Hall. In Bryant's second case, there were symptoms referable to the gastro-intestinal tract, but a state of senile dementia existed and actual dysphagia was not reported: such symptoms as were observed might have been due to the chronic gastric ulcer which was found at autopsy. In this analysis, out of 49 Œsophageal myomata only 3 were believed to have been the cause of such a degree of dysphagia that asthenia and death resulted, and 2 of these were pedunculated.

The precise origin of the tumour was noted in 14 cases, 8 being from the longitudinal muscle coat, 4 from the circular muscle, and 2 from the muscularis mucosæ. In the other cases the site of origin was not recorded, but 7 were said to be covered by the mucous membrane and 1 by the longitudinal muscle.

Generally speaking, the tumours tended to project slightly into the lumen of the Œsophagus.

The smaller growths have a smooth surface and are freely movable; the larger tend to be more nodular. The consistency is firm, and the section shows the greyish-white watered-silk appearance typical of myomata elsewhere. They are usually comparatively avascular, and show up well against the surrounding brownish muscle, but a few are better supplied with blood-vessels and have a pink colour. The tumours are well circumscribed, but a connective-tissue capsule thick enough to be visible to the naked eye is rare. Anitschkow states that only the larger tumours are encapsulated. This is refuted by Tschlenow, who cites his own cases in proof and further denies that the age of the tumour can be made out from its macroscopical appearance or histological structure.

Several cases had associated changes in the Œsophagus. J. D. Stewart reports in a recent article 2 cases of myomata with diverticula, and areas of mucous membrane resembling gastric mucosa. He believes that the diverticulum is due

to the force of swallowing acting on the tumour, causing it to rotate and enlarge eccentrically, and to a thrusting out of the mucosa. Other factors may be a thinning of the wall in the region of the tumour and a congenital defect in the wall. One would imagine that the presence of the myomata in the wall of a muscular tube would lead to some interference with the muscle, but both hypertrophy and dilation are very rare, and were found in only 3 instances. Joseph Meyer reported both dilation and hypertrophy above a large tumour (8 cm. long) which had displaced the common carotid and subclavian arteries to the left, the trachea and innominate artery to the right, and was compressing the left recurrent laryngeal nerve. It may safely be presumed that such a tumour had caused some obstruction, and as no history of dysphagia was recorded, it is probable that the degree of hypertrophy had been sufficient to outcome it. Bezza noted a slight muscular hypertrophy above and below the small pea-sized tumour in his third case, although no obstruction could be demonstrated. In that recorded by Tonoli, a pedunculated hazel-nut-sized myoma was found with a bullous dilatation above it in the upper half of the œsophagus. It was associated with symptoms of dysphagia and vomiting, but no hypertrophy was reported.

Microscopical Examination.—The characteristic microscopical picture is that of myomata elsewhere, bundles of smooth muscle fibres running in all directions; these neoplastic fibres are indistinguishable from those in the muscular coats except for a slight increase in size and their irregular arrangement. The nuclei are rod-shaped, but in transverse or oblique sections appear rounded or oval. The connective-tissue content of these tumours varies considerably in amount. Some, particularly the larger and well-encapsulated ones, show much collagen, and this may even be arranged as fibrous trabeculæ in the nodular type, but in the pedunculated tumours elastic tissue is frequently noted. Anitschkow, in his article, describes a fine collagenous network between each individual fibre, and Bezza reports a fine reticulum in his cases. In the great majority there is a comparative avascularity, and Bezza points out that many of these have both thin-walled vessels and vascular lacunæ lined with endothelium.

Genesis.—Various theories have been advanced as to the origin of these tumours, but it cannot be said that they are very impressive. Tschlenow regards them as being in all probability congenital; while Anitschkow favours the theory of local muscular hypertrophy. Bezza combines these views, and divides the myomata into two groups: (a) Those arising independently of the muscular coats; (b) Those which arise in a muscular coat. The first of these appears to be due to some congenital defect, a misplaced myogenic cell or cells, which in later life proliferated to form the growth; while the latter is ascribed to an over-growth of existing muscle. Bauer noted numerous capillaries at the edge of the tumour, often with a perivascular leucocytic infiltration, and formulated the extraordinary theory that these leucocytes could gradually change into connective tissue and muscle cells, and quoted Kleinwachter as his authority. The absence of leucocytic infiltration in the centre of the tumour was due to the fact that they had already changed into the muscle cells. Scalone, not to be outdone, thought these tumours arose from eosinophils, and believed he had seen transition forms between an eosinophil and a smooth muscle cell! But the etiology of myomata of the œsophagus is as uncertain as that of myomata elsewhere.

Discussion of the Case Reported.—The case reported here is a straightforward one except for the unusual distribution of the hypertrophy. The tumours were small, pea-sized, and situated at the junction of the upper and middle thirds of the œsophagus. They bulged forwards and encroached on the lumen to a considerable extent, reducing its circumference to 2·7 cm. (normal being about 4 cm.). That this had caused some obstruction cannot be denied: there was dilatation and hypertrophy above them, and the firm inelastic nature of the tumours, by limiting the normal distensibility of the organ, was no doubt a contributory factor in the obstruction. But it is difficult to understand why there should be present a gradually increasing muscular hypertrophy and narrowing of the lumen from the site of the tumours to the cardiac orifice. These considerably exceed the changes at the level of the growths and above it: the lumen reaching a minimum circumference of 1·8 cm. just above the cardiac orifice. The degree of hypertrophy below the tumours was proportional to the narrower lumen, and the circular muscular coat at the lower level measured 0·7 cm., compared with a normal of about 0·1 cm. and the 0·2 cm. found immediately above the level of the growth. This has been recorded once before. In other parts of the intestinal tract the hypertrophy associated with narrowing of the lumen is proportional to its duration and extent, and the distal part of the tube, far from showing the hypertrophy so marked in this case, is either of normal thickness or may even show muscular atrophy if the blockage is severe. The only exceptions to this general statement are the case now described, and Bezza's third, which showed slight hypertrophy above and below a small tumour. Bezza offers no explanation of this unusual finding; he merely mentions it in describing the œsophagus. The hypertrophy below may have been a coincidental lesion not directly related to the tumours above it, but the usual causes of hypertrophy are here absent. We can eliminate obstruction from without, and cicatricial stenosis from chronic peptic ulcer or swallowed caustics or acid. Hypertrophy of the lower end of the œsophagus is most commonly associated with dilatation, as in achalasia of the cardia. In this case, no dilatation was present in the lower half, and achalasia, compensated or otherwise, can probably be excluded. The suspicion that this hypertrophy is directly related to the tumours above it is supported by the absence of any other etiological agent.

It is possible that these tumours in the middle of the œsophagus directly affected its nervous mechanism, as they lay in close proximity to Auerbach's plexus, and they may well have caused irritation and irregular muscular contractions. It is also possible that, replacing, as they did, a large part of the circular muscle coat, muscular conduction was impeded and blocked, and the distal hypertrophy was the result of irregular and uncontrolled contractions. On the other hand, in squamous carcinoma of the œsophagus, where the possibilities of local muscular and nervous interference are much greater, one does not get hypertrophy below the obstruction.

In conclusion, one may mention yet another possibility—idiopathic hypertrophy of the œsophagus. In view of the advance of diagnostic methods, it is now a very rare lesion, but cases confirmed by post-mortem study are described (Wood). It may be that here we have multiple congenital abnormalities, as a congenital origin has been suggested for both myomata of the œsophagus and the so-called idiopathic hypertrophy.

SUMMARY

A case of multiple myomata of the œsophagus which resulted in fatal stenosis is described, and the literature is briefly reviewed.

I wish to express my sincere thanks to Dr. J. Greig Thomson for his stimulating assistance, to Miss E. A. Richardson for translating the Italian, and to Professor G. Grey Turner for his suggestions and criticism.

BIBLIOGRAPHY

- ANITSCHKOW, *Virchow's Arch. f. path. Anat.*, 1911, ccv, 443.
 BAUER, *Ibid.*, 1917, ccxxiii, 34.
 BEZZA, *Pathologica*, xxiv, 71.
 BRYANT, *Jour. Amer. Med. Assoc.*, 1905, xlv, 2008.
 COATS, *Glasgow Med. Jour.*, 1872, N.S. iv, 201.
 EBERTH, *Virchow's Arch. f. path. Anat.*, 1868, xliii, 137.
 FAGGE, *Trans. Pathol. Soc. Lond.*, xxvi, 94.
 GAJET, *Soc. des Sci. méd.*, 1910, Nov. 9.
 HALL, *Quart. Jour. Med.*, ix, 409.
 ROLLESTON, *Trans. Pathol. Soc. Lond.*, 1, 73.
 SPITZNAGEL, *Radiology*, 1931, xvii, No. 3, 570.
 STEWART, *Arch. of Pathol.*, xii, 77.
 TRASTOUR, *Bull. Soc. anat. de Nantes*, 1881, 7.
 TSCHLENOW, *Virchow's Arch. f. path. Anat.*, 1923, ccxlii, 239.
 WOOD, *Arch. of Pathol.*, xiv, 767.

The Following Papers were Read in Abstract only :—

- FRANK, *Inaug. Dis.*, Munchen, 1911.
 ILLIG, *Inaug. Dis.*, Giessen, 1894.
 MAUCHER, *Inaug. Dis.*, Zurich, 1900.
 MEYER, P., *Inaug. Dis.*, Freiburg, 1887.
 MEYER, J., *Inaug. Dis.*, Munchen, 1882.
 REHER, *Inaug. Dis.*, Kiel, 1885.
 PICHLER, *Prag. med. Woch.*, 1897, xxii, 445.

TRAUMATIC RUPTURE OF THE URETHRA EIGHT PERSONAL CASES, WITH A REVIEW OF 381 RECORDED RUPTURES

(CHILDBIRTH AND WAR INJURIES EXCLUDED)

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TRAUMATIC rupture of the urethra is an uncommon injury. Some figures show that only 4 cases per 1400 in-patient admissions are to be expected in any one year; yet even this may be a too generous proportion, for many surgeons pass through a full professional life without meeting more than a single example. Familiarity with their management, however, is desirable, for hesitant or defective treatment may plunge a healthy life into one of strangury, stricture, sinuses, stone, perineal abscesses, or other miseries of urinary disease.

Refreshment is sometimes gained by recounting individual experiences. Eight personal cases are submitted, each possessing some separate point for discussion or improvement. In addition, all the available writings of the past twenty years, yielding some 381 cases, have been surveyed in order to present a reliable picture and to measure the trend of modern urological views.

Details of the eight cases will first be given, but comment on their treatment will be reserved for the general discussion. An analysis of the total cases will contrast 'bulbar' and 'membranous' injuries, and show how often the perineum has been incised, the torn ends sutured, in-dwelling catheters or suprapubic drains used, and the number of subsequent strictures recorded. The discussion will indicate the debatable points for support or condemnation, and will suggest the safest method of negotiating the emergency.

AUTHOR'S CASE REPORTS

Case 1.—R. M., aged 70 (July 27, 1933), fell while standing on a box with legs astride the edge. Immediate sharp perineal pain, and fainting.

On admission half-an-hour after the accident there was extensive bruising of perineum and groins; local tenderness under pubic arch; blood dripping from the urethra; intense desire but inability to pass water. Warned not to micturate.

Immediate suprapubic cystostomy. A retrograde gum-elastic catheter was passed down to the rupture in the bulb, and after gentle rotation its eye emerged at the penile meatus. A soft rubber catheter was tied to the bladder-end of the gum-elastic and by pulling on the penile end the rubber catheter was piloted down the urethra until only the tip rested inside the trigone. This rubber tube now became an indwelling catheter. A large suprapubic drain was then inserted and connected to a Sprengel's pump.

The in-dweller was left out for twenty-four hours on the 28th day, when the patient passed small amounts of urine through the penis, and through the healing suprapubic wound. The latter finally closed on the 35th day.

Urethroscopy seven weeks after the accident, with the added advantage of Mr. V. E. Lloyd's expert eye, showed a small ledge of scar tissue on the floor of the bulb, but no sign of a stricture. A 26 F. passed easily. Unfortunately three days after this examination a small indurated swelling 4 in. by 2 in. appeared along the closed suprapubic scar. Catheterization

produced a foul blood-stained urine and much gas. The cystostomy was re-opened and bladder wash-outs were maintained for five days. The wound re-healed on the 20th day. The patient was finally discharged exactly three months after the accident, "passing water freely".

EXAMINATION 2½ YEARS AFTER THE INJURY.—Health excellent; no catheterization since discharge from hospital; no pain or difficulty in micturition other than occasional dribbling and frequency—attributable to an enlarged prostate obvious on rectal examination. There is no palpable evidence of urethral scar and the stream is good and strong. The patient is emphatic that the penis has atrophied since the operation and when questioned about erection maintains that "before the injury it used to fly out at any time, but now it is only proud when the bladder is full, and then not very strong". His age, however, must be taken into consideration. Urethrogram shows a slight deformity at the site of injury but no stricture. The membranous urethra is elongated and irregular. (See Fig. 216.)

Case 2.—A. B., aged 25 (Aug. 25, 1933), was standing with one leg on a table and the other on a step-ladder when the latter slipped away. One limb shot horizontally along the table, the other vertically downwards, allowing the perineum to fall heavily on the edge. The patient lay in great pain for twenty minutes, but then walked alone to hospital.

On admission half-an-hour after the accident there was intense perineal pain and a tender lump on the right side of the bulbous urethra, but no ecchymosis. Clear urine was unfortunately voided in the surgery for testing, but later when wishing to pass water the patient only bled from the meatus.

Immediate operation. A catheter was passed and, although there was a slight hitch in the bulb, the instrument slid comfortably into the bladder, indicating only a partial rupture. The bladder was opened to divert the stream. A new catheter with several lateral holes was passed up the penis to act as an in-dweller, and, with a finger down the suprapubic incision, the tip was made to lie just below the internal sphincter to avoid any trickling of urine along the catheter to the traumatized area. A Sprengel's pump was attached to the cystostomy drain.

The urethra was irrigated daily and lightly through the multiple holes of the in-dwelling catheter. On the 11th day there was slight difficulty in changing the catheter, and pus suddenly poured from the urethra. Culture yielded *Streptococcus* and *Proteus*, but no *B. coli* or *gonococcus*. Wassermann and Kahn tests were negative. On the 15th day the catheter lay out on the bed all night, and as it could not be returned the suprapubic incision was re-opened by a locum. A descending catheter readily appeared at the meatus, and a soft rubber catheter was left in the urethra. The patient lost ground, the temperature remained at 103°, and a painful, soft, fluctuant, and very tender perineo-scrotal swelling appeared. A mid-line incision produced thick reddish pus, and the temperature became normal two days later.

Eight weeks after admission the catheter was out for one hour, and on the next four days for two, four, twelve, and twenty-four hours. The patient left hospital nine weeks after the injury, passing water quite well. A urethrogram looked normal, but on passing a sound there was a definite soft stricture which yielded to kindly dilatation. This was repeated once a month until the eighteenth month.

EXAMINATION 2½ YEARS AFTER THE INJURY.—The patient looks remarkably well, but although he never has to get up at night he cannot hold his water for longer than one hour by day. There is a small hard lump with a tiny depression proximal to it in the perineum. The stream is good and thick, with plenty of force; the urine is normal. At first the penis bent down on erection, but soon straightened out. Coitus still produces intense perineal pain which lasts until the following noon, and is only indulged in from time to time. Urethrogram now shows a good wide channel with some irregularity, but no stricture, in the bulb. (See Fig. 216.)

Case 3.—J. K., aged 5 (April 15, 1932), was knocked down by a car, the wheel passing over the pelvis.

On admission half-an-hour later the boy was very shocked and in great pain. The lower abdomen was intensely bruised, swollen, rigid, and very tender. There was a large gap between the two halves of the symphysis pubis. Blood oozed from the meatus, but there was no perineal hæmatoma. In spite of the critical condition of the child, delay seemed fraught with the risks of extravasation, of severe intestinal injury, and the onset of secondary shock.

An immediate subumbilical incision exposed large blood-clots in the abdominal wall. The peritoneal cavity was deliberately opened, and as a rapid examination of the abdominal viscera showed no injury, the cavity was closed. A distended bladder was noted riding up towards the umbilicus, attached by two lateral folds of peritoneum containing the vesical vessels, almost like a toy balloon tethered by two convergent strings. There was an ominous gap of at least three inches between the right and left parts of the symphysis, and through the gap the apex of a miniature prostate could readily be palpated. The bladder was emptied by puncture with a large-bore needle attached to a sucker to avoid soiling, and opened. A soft rubber catheter was retrograded to the rent in the membranous urethra, the eye seized by forceps, and easily pulled up through the gap. The tip of an ascending catheter from the penis was similarly seized and elevated (*Fig. 215*). The tubes were joined, and by pulling on the meatal guide the descending catheter was drawn along the urethra to appear at the glans. This in-dweller traversed a gap of two inches between the torn urethral ends. Light digital pressure on the trigone easily pushed the bladder downwards and forwards to approximate these torn ends, but the viscus rose again on removing the finger. One regretted that something could not be devised to hold the bladder down. It was clear that a suture could readily be inserted into the accessible prostatic end, but the penile end was so deeply situated, and surrounded by such traumatized tissue, that it was doubtful whether the suture could be passed into it, or secured sufficiently to keep the torn ends together. Insertion of the suture would take time even with the advantage of the enormous symphyseal gap, and was abandoned as the child was too ill for much manipulation. A perineal section was also out of the question—it could be attempted later if necessary. Accordingly the abdominal incision was hurriedly closed, leaving a bladder drain for later attachment to a Sprengel, and a long strip of corrugated rubber to drain both the cave of Retzius and the vast traumatic cavern below it. After a blood transfusion the boy was returned to bed.

The next few days were made pleasant by his splendid recovery. He was as yet too weak and his pelvis too shattered for the adoption of the Fowler position. Obviously sitting him bolt upright would encourage the abdominal viscera to force down the bladder; on the other hand, high blocking of the bottom of the bed would ensure all urine falling into the fundus from the ureteric orifices, and thus prevent any trickling along the resident catheter into the traumatized area. A continuous warm saline irrigation up through the catheter and out through the suction tube was an attempt at compromise—in the hope that any leak of urine would be so dilute as to rob it of any of the toxic effects familiar in extravasation. Unfortunately, on the 12th day pus oozed from the Retzius drain, but in spite of this the suprapubic tubes were removed on the 21st day. Owing to the gross separation of the torn urethral ends, the in-dwelling instrument was left in until the sixth week.

The boy now passed urine from the penis, and through the small purulent suprapubic fistula. Removal of a sequestrum at the eighth week terminated a short-lived osteomyelitis, and the extraction of two small bladder stones through the dilated fistula cleared up any pus from that source. At the second lithotomy the fistulous track was found adherent to the back of the pubis, so it was dissected off the bone. The fistula, which had remained open for nearly six months, closed in a week!

The tenth month saw the patient discharged. His wound was soundly healed, micturition was free, uroselectan showed no abnormality of the urinary tract, and urethrography demonstrated good canalization of the whole urethra. Yet he disliked the passing of a No. 6 catheter—a feat only possible in the hands of Sister Dobson, to whom great tribute is due for so completely winning the confidence of the child and his urethra throughout the hospital catheterizations.



FIG. 215.—*Case 3.* Showing the catheters being pulled up through the gap between the two halves of the fractured symphysis pubis. The tips were united and the bladder catheter pulled down along the urethra to act as an in-dweller. By this simple means a perineal incision was avoided.

Urethroscopy at the eighteenth month, again with Mr. V. E. Lloyd, showed a well-defined circular stricture in the membranous portion, but a No. 6 English—the largest his penis would allow—slipped into the bladder without difficulty.

EXAMINATION 4 YEARS AFTER THE INJURY.—The patient is perfectly well; there is no pain or difficulty with micturition; the stream is good and strong; the urine clear. There appears to be no atrophy of the penis. Uroselectan normal. Urethrogram as shown in *Fig. 216*.

Case 4.—D. C., aged 47 (Aug. 29, 1934), was loading logs from a ship to a barge. One trunk swung over on a crane and hit the patient on his pubis and penis, knocking him from the vessel into the bottom of the barge. He experienced great pain at the tip of his glans, and to a lesser extent in his pelvis. He could not rise, and was brought to hospital twenty minutes after the accident.

On admission he was in extreme shock and bleeding freely from the penis. There was intense pain in the lower abdomen, which was rigid, and in both legs. A large hæmatoma of the perineum rapidly developed and extended to the scrotum. X rays showed fracture of both sides of the pubis and a fracture-dislocation of the right sacro-iliac joint. A catheter was passed in the surgery and although buried to the hilt merely drew off blood. On running in a measured amount of fluid a very little returned. The patient was admitted to the wards, intravenous therapy commenced, and an immediate operation performed.

A subumbilical incision exposed much blood-clot and a distended bladder riding out of his pelvis, rather higher than normal. The extreme dislocation seen in *Case 3* was not present. A fragment of bone projected into the bladder wall and was causing much hæmorrhage from the vesical venous plexus. The bleeding points were controlled, and the incised viscus was found to contain blood. The cave of Retzius opened into a vast cavern at the apex of the prostate; blood welled out of this chasm, but could not be traced to any particular bleeding points. Catheters were rapidly retrograded and 'antegraded' to the rent in the membranous urethra, their tips were seized, brought up through the wound, united, and the upper one pulled down along the urethra by traction from the penile one. A finger exploring the ruptured site found a long gap between the torn ends traversed by the catheter. Digital pressure on the trigone easily approximated these torn ends, but, as in *Case 3*, the viscus rose again on removing the finger. A piece of thick rubber sheeting was cut into a florin-sized circle with a hole in the middle to admit the shaft of a stout De Pezzer catheter. This improvised 'extension-catheter' was attached to the already resident sound, and 'rail-roaded' down the urethra until the head and collar rested upon the trigone (*Fig. 217*). Light digital traction on the penile end of this new in-dwelling catheter readily held the bladder down and obliterated the gap between the torn ends. The whole procedure

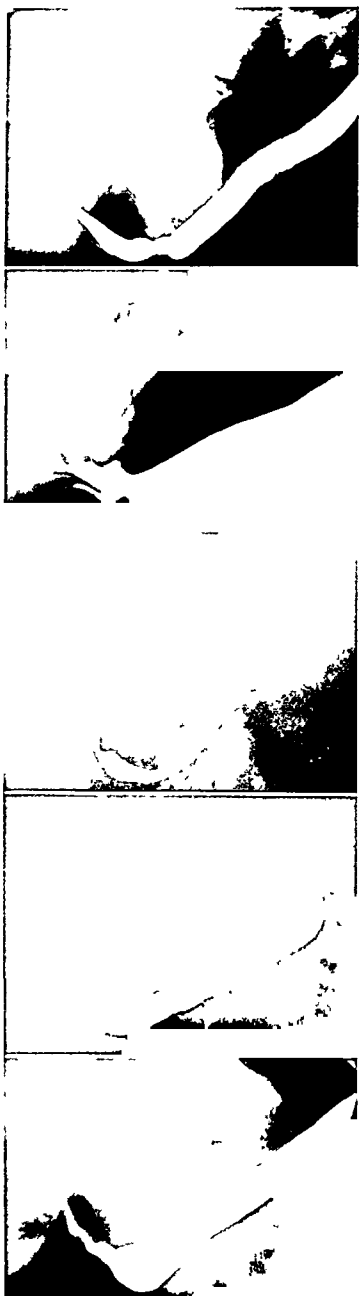


Fig. 216.—Urethrograms of *Cases 1, 2, 3, 4, and 5* (from above downwards).

occupied only a few minutes. A large-sized suprapubic tube was placed in the bladder, and the patient returned to bed. A Sprengel was attached to the suprapubic tube, and a 1-lb. weight to the penile catheter by means of a pulley over the end of the bed. One felt that such a pressure was considerably less than that of the various forms of hydrostatic bags, especially those fitted with strong springs clamping the inside of the bladder to the perineum.

A visit to the ward late that night revealed a patient who affirmed that his penis and perineum were remarkably comfortable. His condition was excellent. The pound pressure was maintained for twelve days, and throughout this time there were repeated denials of any discomfort. On the 4th and 12th days, at the changing and removal of the collared De Pezzer, a cystoscopic light placed down the suprapubic tube-shaft showed a trigone bearing no marks of the slightest pressure necrosis.

[The author's tenure of office at the Hospital now terminated, and thanks are due to the authorities for further details.]

Slight urinary sepsis yielded to bladder wash-outs. On the 28th post-operative day sudden profuse bleeding per rectum occurred, and was repeated next day. Blood transfusions were given. A radiograph showed the right sacro-iliac joint displaced. On the 50th day a fistula suddenly appeared on the under surface of the penis just behind the glans, and a second one "in the left groin close to the base of the scrotum". The appearance of these fistulae at such a late date was presumed to be due to peri-urethral abscesses. The groin fistula rapidly closed, and the patient was discharged after five months. The refractory penile fistula occasioned four attempts at closure.

EXAMINATION 18 MONTHS AFTER THE INJURY.—The patient is very frail, and progression with two crutches is slow and difficult. He has much pain in the back and hips. There has been no bougie treatment since discharge from hospital, and there is now a good urinary stream, but pain at the end of the penis on micturition. Frequency: day, every five minutes; night, 3 to 4 times. The urine contains many phosphates, but no albumin or pus. The prostate appears normal. There have been no erections, and the patient states that his penis has atrophied since the accident. Yet the organ looks of normal proportions. Urethrogram shows an enlarged canal behind the corona, but no stricture or elongation of the membranous urethra. (See Fig. 216.)

Case 5.—F. H., aged 48 (July 26, 1933), skidded from his bicycle under a charabanc; dazed; brought to hospital within an hour.

There was extreme bruising and effusion from above Poupart's ligament down the inner side of the right thigh. No perineal tenderness. No blood could be expressed from the urethra; there was no evidence of intraperitoneal effusion or rupture. The pelvis was fractured—both ischio-pubic rami were separated from the symphysis. Catheterization produced clear urine with only a few microscopic red cells.

Two days later the extensive thigh effusion was tapped: 300 c.c. of blood-stained fluid were withdrawn, and found to contain 0.81 per cent of urea—obviously urine. A groin drainage was established, but as the patient appeared to have no difficulty in micturition it was deemed inadvisable to do more. A pyrexia of 101° – 103° , and an occasional drop of urine from the groin, still persisted, so on the 15th day the bladder and urethra were X-rayed. The cystogram appeared normal, but urethrography showed a track of lipiodol extending from the membranous urethra to the groin.

An in-dwelling catheter was inserted, but caused acute pain and restlessness, so was removed. The groin sinus rapidly cleared up, and apart from an unexplained sharp rigor

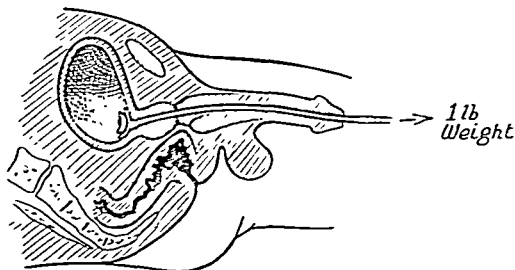


FIG. 217.—Case 4. Showing the de Pezzer catheter with its circular rubber collar in position, after it had been pulled down the urethra by means of the manoeuvre employed in Case 3 (see Fig. 215). A 1-lb. weight attached to the catheter pulled the trigone downwards to approximate the torn ends. A perineal exploration and end-to-end suture could not have been performed as the man was too ill, and this improvised method was found to work well. The patient always maintained that he was comfortable, and 'cystoscopy' down the suprapubic tube-shaft on the 4th and 12th day showed no trace of pressure necrosis. It was felt that the 1-lb. weight was considerably less than that of various forms of hydrostatic bags or strong springs used to press the prostatic bed against the perineum.

at the end of five weeks, and pyrexia for the next seven days, the urinary convalescence was good. Owing to his fractured pelvis the patient remained in hospital for eleven weeks.

EXAMINATION 2½ YEARS AFTER THE INJURY.—Has not experienced pain or difficulty with micturition: day, 4 hours; night, nil; no sex disturbance. Urethrogram normal. No sign of any fistulous tract or stricture. (See Fig. 216.)

Case 6.—W. G., aged 42 (April 2, 1932). His van was struck by a skidding lorry, and he slipped down between the shafts. His horse kicked the end of his penis.

On admission twenty minutes later the patient was extremely shocked. The glans penis was a most gruesome sight. Swollen to the size of an orange, it was plum-coloured and dripping with blood. There was a deep T-shaped laceration, the horizontal bar running around the corona and partially severing the glans from the shaft, the vertical bar completely laying open the distal part of the urethra up to the meatus. The remainder of the penis was much bruised.

Immediate operation. Toilet. Suture of urethra in two layers around a self-retaining catheter, and of the glans to the shaft. Sprengel's pump to catheter.

All the sutures held except a few on the under surface near the external meatus. The

date of removing the in-dwelling catheter was unfortunately not recorded, but the patient left hospital on the eighteenth day with a healed wound.

EXAMINATION 4 YEARS AFTER THE INJURY.—There has been no instrumentation since confinement in hospital, but the water very occasionally burns; day, 3 hours; night, once. Micturition is accompanied by much spraying of the stream, for there is a slight deficiency of the right extremity of the penis, with a very patulous external opening. On erection the glans balloons out to the left, pulling the frenum very taut and causing much pain. Ejaculations are "sideways". Coitus is only undertaken once in two months. There is no stricture.



FIG. 218.—Case 7. Showing the enormous perineal fistula.

the development of a stricture in the next six months. Repeated dilatations have since been necessary. At the age of 14, patient consulted a French surgeon, who diagnosed tuberculous epididymitis and gave injections, strongly smelling of creosote, into the testicle. Almost immediately afterwards "water" appeared in the right knee. A radiograph was negative. The limb was splinted and the patient soon returned to cricket, ski-ing, etc. At the age of 16, while at winter sports, the stricture again became troublesome. A peri-urethral abscess developed, and was opened and drained by a Swiss surgeon. A mid-line perineal catheter was inserted into the bladder behind the abscess. Tests for tuberculosis again were negative.

Last year the patient, aged 27, consulted the author for a now definite tuberculous knee and for a bulbar stricture with a long, irregular, and gaping perineal fistula behind it, through which urine sprayed on micturition (Fig. 218).

As the knee showed active disease it was considered the most urgent matter, and the patient was sent to an orthopaedic surgeon. An excellent excision has produced a stable limb.

The urethra is now under consideration. The definite traumatic origin after cystoscopy long before the advent of suitably small children's cystoscopes, the non-advancement of the fistula during all these years, and the examination of the edge, all rule out tubercle as the source of the trouble.

Case 7.—X. Y., aged 27. Albumin in urine at the age of 12. Cystoscopy was said to be essential to eliminate tuberculosis. This was attempted, but followed by much urethral bleeding and

The stricture itself and the elongated perineal fistula present separate problems. A suprapubic deviation preceding any operative measures by at least two weeks will be a necessary and safe preliminary. Cutaneous autoplasmic flaps, such as Fruchaud's, are fortunately unnecessary here, and should only be used as a last resort where mobilization fails. Most of such flaps grow hairs.

Case 8.—A. D., aged 40 (May 7, 1930), stated that he caught his penis on his pyjama strings when getting out of bed one hour before admission. There was profuse bright bleeding from the urethra and a tender spot just behind the glans. Ice-bags were placed above and below the penile shaft, and a large dose of morphia administered. Cessation of hæmorrhage followed in twelve hours, and discharge from hospital in two days.

SIX YEARS AFTER THE ACCIDENT.—There has been no difficulty in passing water, and no catheterization has been necessary.

CONCLUSION.—Rupture of the mucous membrane of the pendulous urethra : of doubtful cause.

Table I.—SUMMARY OF PERSONAL CASES

Case Number ..	1	2	3	4	5	6	7	8
Age (in years) ..	70	25	5	47	48	42	12	40
Site ..	Bulb	Bulb	Memb.	Memb.	Memb.	Penis	Bulb	Penis
Cause ..	Astride	Astride	Car	Fall	Fall	Kick	Surgical instrument	?
Fractured Pelvis ..	—	—	I	I	I	—	—	—
Symptoms :—								
Shock ..	I	—	I	I	I	I	—	—
Pain ..	I	I	I	I	I	I	—	I
Bleeding from penis ..	I	I	I	I	No	I	—	I
Perineal tumour ..	—	I	—	I	No	Penile	—	Penile
Perineal tenderness ..	I	I	I	I	No	Penile	—	Penile
Ecchymosis ..	I	—	—	I	I	Penile	—	—
Retention* ..	I	P	—	—	No	—	—	—
Extravasation ..	—	—	—	—	I	—	—	—
Treatment :—								
Suprapubic diversion	I	I	I	I	—	—	—	—
In-dwelling catheter ..	I	I	I	I	P	I	—	I
Duration ..	1 mth.	2 mth.	6 wk.	3 wk.	1 hr.	?	—	2 days
Perineal section ..	—	—	—	—	—	—	—	—
Sprengel ..	I	I	I	I	—	I	—	—
Complications :—								
Pus in urine ..	I	I	I	I	—	—	—	—
Abscess ..	I	I	osteomyelitis	P	—	—	—	—
Perineal fistula ..	I	—	—	I	—	—	I	—
Stricture ..	No	No	No	No	No	No	Yes, but no treatment	No
Years followed up	2½	2½	4	1½	2½	4	—	6
Impotence ..	PP	Pain	—	I	—	—	—	—
Miscellaneous ..	—	Bladder stone	Bleeding per rectum	—	—	—	—	—
Hospitalization (in weeks)	12	9	40	22	11	18 days	—	2 days

* Most patients brought to hospital "under the hour"; retention therefore unlikely.
P = Presumed. PP = Partial.

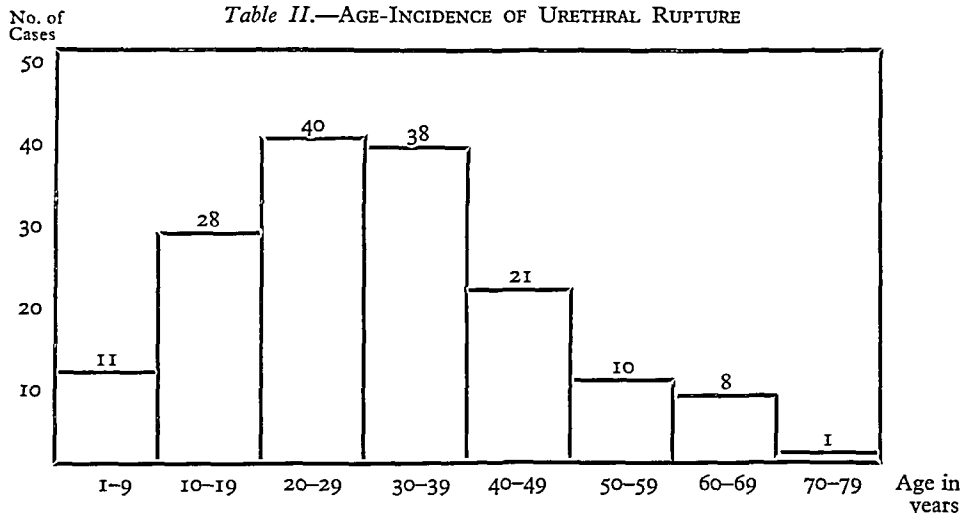
ETIOLOGY, BASED ON 381 RECORDED CASES

The ages, sex, sites, and causes of rupture in the 381 recorded cases are detailed in *Tables II to IV*, cases of rupture of the female urethra being shown in *Tables III and IV*.

AGE

The age is stated in 157 cases; unstated in 177 cases. The remaining 47 further cases comprise 15 "boys", 1 "young student", and 21 "adults".

Table II.—AGE-INCIDENCE OF URETHRAL RUPTURE



SITE

The term 'membranous' in this paper and in the Tables embraces the whole of the posterior urethra from the bladder neck to the junction with the bulb (5 cm.), for at operation it is nearly always impossible to determine the exact site of the rupture owing to the astonishing amount of bruised tissue and blood-clot. Further minute differentiation is of little practical value.

Table III.—SITE OF URETHRAL RUPTURE

	RECORDED	PRESUMED*	TOTAL	PERCENTAGE OF TOTAL CASES
Bulbous	150	37	187	50
Membranous ..	114	42	156	40
Pendulous .. .	19	—	19	5
Female	7	—	7	2
Site unstated ..	—	—	12	3
Total	381	

* Suggested and almost made certain by the other details of each record. In later tables it has been found inadvisable to separate the groups 'recorded' and 'presumed'.

CAUSES

It will be observed in *Tables III* and *IV* how rarely the shaft of the penis (19) or the female urethra (7) is injured, and how responsible is fracture of the pelvis in producing membranous ruptures (107). Falling 'astride' a plank or pole nearly always causes injury to the bulb by driving that portion of the urethra sharply against the cleft of the pubic arch (157). It is remarkable, however, that this method may occasion injury to the membranous urethra (17). Falling heavily 'on the side' without fracturing the pelvis only produces 5 membranous ruptures, but such falls can actually cause bulbar (9) instead of the usual membranous lesions (112).

The list contains a few rare accidents, of which coitus, a string tied round the penis to stop a gonorrhœal discharge, and a solitary case of indirect violence are the most interesting. In the last a man tumbled from a lorry and in mid-air kicked up at a sack falling on top of him.

Table IV.—ACCIDENTS CAUSING URETHRAL RUPTURE

	BULB	MEM- BRANOUS	PENDU- LOUS	SITE UN- STATED	TOTAL	PERCENT- AGE
<i>Falls</i> :—						
'Astride'—e.g., a ladder rung, girder, or grating, edge of barge or buggy wheel, arch of saddle, engine buffer	157	17	—	—	174	46
'On side', without pelvic fracture—e.g., motor cycle wobble, knocked down by car ..	(6 3 (107+5 = 9)	(107+5 = 112)	2	3	13	3
'On side', with pelvic fracture—e.g., crushes, car accidents, falls from buildings ..	—	—	—	—	113	30
<i>Blows</i> :—Direct hits on perineum—e.g., kick from human or horse, sledge-hammer, barrow handle, car handle 'knocking' violently on perineum ³⁵	9	7	—	4	20	5
<i>Instrumental</i> :—Surgical catheter or cystoscope (7) ⁹ ; self-inflicted dement (1); irrigation with lipiodol (2) ⁴⁰ ; with argyrol (1) ⁴⁰ ..	11	—	—	—	11	3
<i>Miscellaneous</i> :—						
Coitus ^{20, 26, 43, 63}	—	—	4	—	—	—
Shot in stooping position ³⁹	—	1	—	—	—	—
Penis whirled off in machine (1) ⁴³ ; twisted (3) ²⁰	—	—	4	—	—	—
String tied round penis ²⁰	—	—	2	—	—	—
Indirect muscular violence ³⁰	—	1	—	—	12	3
<i>Female</i> :—Car accidents with fractured pelvis (5) ⁶⁹ ; blow (1) ⁴⁷ ; coitus (1) ³	—	—	—	7	7	1
<i>Cause unstated</i>	1	18	7	5	31	8
Totals	187	156	19	19	381	

Of the females, the sad state of a young woman of 23 is recorded. An attempt at coitus on her bridal night was followed by severe pain, abundant hæmorrhage, and incontinence, the latter persisting for three long months, during which

time the patient kept mostly to her bed. On examination a finger could be introduced "with great ease and without pressing" up the urethra into the bladder. It is interesting to note that two operations were necessary to close the defect. At the first an in-dwelling catheter was used, but at the second a suprapubic derivation was performed and continence restored.

It is odd that there are only 2 female ruptures in the literature apart from no less than 5 occurring in the practice of Turley; all the five are said to be due to automobile accidents.

The irrigation disasters are included as curiosities; Jules Janet reported them as "ruptures", for each patient suffered a sharp burning sensation, and the penis was swollen and blue-red immediately after the injection. One followed the injection of argyrol 20 per cent, one lipiodol for urethrography, and another lipiodol for chronic stricture. These are happily the only three such records during the past twenty years, and it is singular that they are all reported by the same author.

TYPE

The type of urethral rupture depends on the degree of damage inflicted.

In *partial* ruptures either the mucous membrane alone is torn, as in penile injuries (e.g., Case 8), or more commonly part of the tube may be split across (e.g., Cases 1 and 2).

In *complete* ruptures a blow sufficient to sever the tough urethra usually causes much crushing of the ends, and the gap thus formed is further increased by the retraction of the elastic tissue.

'*Compound fractures*' of the urethra arise as a result of severe perineal laceration by blunt instruments. One intoxicated man who had fallen asleep on the road was carried some distance by the starting-handle of a car "knocking violently on his perineum".

SYMPTOMS AND SIGNS

The symptoms of traumatic rupture are few and straightforward, and, except when submerged in grosser pelvic damage, readily lead to a correct diagnosis. This is reflected in the short consideration given to symptoms in the recorded cases—in well over three-quarters of the total 381 these details are omitted altogether. Shock, pain, bleeding from the penis, perineal hæmatoma and ecchymosis, abnormality of micturition, and occasional extravasation, form the principle symptoms.

Shock is severe even when the extreme end of the penis alone is injured (cf. Case 6).

Pain may be absent on account of the shock, or obscured by the agony of a pelvic fracture. In uncomplicated cases it is usually felt in the perineum, but may radiate to the tip of the glans.

Bleeding from the penis is often stated to be more profuse in partial than in complete ruptures, for it is maintained that the wide and irregular separation of the torn ends prevents the escape of blood down the normal urinary channel (Legueu). The series shows this observation to be of more theoretical than practical value, for several cases with considerably retracted ends poured with blood. This even occurred where enormous reaches of the pelvic floor had been

torn up and gaping chasms had been filled to overflowing with bloody fluid (cf. Cases 3 and 4).

Table V.—SYMPTOMS OF URETHRAL RUPTURE

SYMPTOMS	No. OF CASES*
Retention	95
Penile bleeding	69
Perineal hæmatoma or tumour	73
Perineal ecchymosis	45
Perineal or penile pain	36
Extravasation of urine	35
Shock	27

* These figures are very small, for in over three-quarters of the 381 cases no symptoms were recorded.

Perineal hæmatomata have been tied down to rigid anatomical planes—and are affirmed to occur only in ruptures below the triangular ligament. Such hæmatomata are governed more by the severity of the injury, and by the lapse of time, than by anatomy. A crush or fall sufficient to fracture a pelvis, or indeed a tough urethral tube, is not likely to respect fascial boundaries, and blood may fill any nascent cavity or gravitate to the perineum. Wheeler is one of the few who have emphasized the possibility of a perineal hæmatoma being due to a tear above, as well as below, the barrier of the triangular ligament. Again, cases not seen for two to even seven days will have had ample time for blood to collect in the dependent parts.

Case 4 had no hæmatoma when examined in the surgery, but an incredible perineal tumour was present on arrival at the ward. This had actually overflowed to the scrotum by the time the patient was on the operating table—only about an hour after the injury.

Another type of hæmatoma present in membranous ruptures, and very seldom stressed, is the soft boggy mass which can be felt bulging into the rectum and occupying the place of the prostate when the latter has been wrenched from its bed (Fig. 219).

Ecchymosis may be expected in all blows or falls astride where direct violence in inflicted on the perineum, yet it is also found in posterior injuries. Of 45 recorded cases 28 were bulbous and 17 membranous, but as 6 of the latter were due to falls astride, and therefore to local bruising, the correct proportion might be interpreted as 34 direct to 11 indirect injuries. It is interesting to note that the solitary case of indirect violence, i.e., kicking up at a falling sack, and only 3 of the author's series (2 membranous, 1 bulb), presented this perineal staining.

Abnormality of micturition: The term 'retention' of urine has been too widely used to embrace all urinary difficulties. At the one end of the scale patients have been able to pass water fairly well for days, and then real retention has gradually

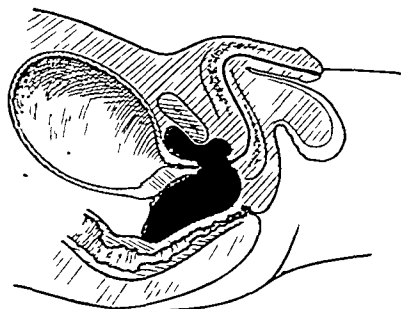


FIG. 219.—Shows the large hæmatoma which can often be felt as a boggy mass per rectum.

overcome them. All such cases are not limited to partial ruptures. Smith and Mintz record a remarkable case of complete separation of the torn urethral ends where the patient passed water for six weeks before complete stoppage occurred. Barnett records cases of 13 days, Morison 10 days, and Jastram 8 days before cessation. Flandrin records a case of a patient who fell astride, continued work, went to bed without voiding, passed a good night, and was harassed at not being able to micturate the next morning. This is quite a common experience. Intermediate cases have painful and frequent passage of blood-stained urine.

At the other end of the scale there is sudden and absolute retention which is intensified by frequent wild attempts to void, and sometimes rewarded by the squeezing out of a few drops of blood. In these cases full bladders can usually be palpated provided the lower abdomen is not thrown into spasm by fracture of the pelvis.

Of the meagre symptoms supplied by the records, "retention" or "inability to void" seem to have impressed the writers the most.

Extravasation of urine should seldom occur if a diagnosis of rupture is made early enough. The bladder sphincters fortunately nearly always escape, for they usually lie above the site of rupture; a protective sphincteric spasm can thus prevent the soiling of the injured field. When extravasation occurs, however, there is a strong tendency for the tissues rapidly to necrose, and for the penis, scrotum, and perineum to become œdematous, blue-black, and dotted with greyish sloughs or wholesale gangrene. Hamilton Bailey and Huddy state that 22 per cent of traumatic ruptures have extravasation, and when this occurs the mortality increases to 20 per cent.

DIAGNOSIS

The passage of a catheter is the most unreliable means of diagnosis, and this should not be employed. Numerous examples of the false information it gives are to hand. Young chides himself for not operating on a patient whose bladder could be so easily catheterized that a membranous rupture was missed until a fatal sepsis allowed the true condition to be revealed post mortem. Wheeler delayed operating upon a ruptured bladder for twenty-four hours because repeated catheterization withdrew a full quantity of blood-stained urine. At operation he was able to demonstrate that the sound had passed through the rent in the bladder into the urine-laden peritoneal cavity.

There are no less than 42 cited occasions when diagnostic catheterization was unsuccessful. In only 6 cases was this manœuvre definitely forbidden.

In the rare accident of double rupture, bladder and posterior urethra, operation alone (or autopsy) discloses the dual injury—e.g., Berry's Case 2; Peacock and Haines's Case 1 (died) and Case 3 (16 operations for repair).

Lipiodol urethrograms sometimes are of use in diagnosing doubtful cases—e.g., the author's Case 5, Partsch and Breitländer's Case 4.

Air injected up the penis has been used by Vaughan Reid to differentiate urethral and bladder injuries. He possesses pictures of air passing out of cystostomy rents and bubbling up between the coils of small intestine. In an article in preparation the author shows that even oxygen is not absolved from the remote danger of air embolus.

COMPLICATIONS AND CONCOMITANT INJURIES

Table VI conveys a good idea of the various types of complications that are to be expected, and beyond a consideration of the most serious difficulty—*stricture formation*—no further allusion to them will be made.

Table VI.—COMPLICATIONS AND CONCOMITANT INJURIES IN RUPTURED URETHRA

LESION						No. of Cases
Stricture	100
Sepsis :—						
Wound	70
Urethral fistula	37
Periurethral abscess	13
Osteomyelitis of pelvis	2
Intestinal fistula	2
Stone : Bladder (4) ; Urethra (3)	7
Second and subsequent operation (one of these cases had 16 operations)	40
Epididymo-orchitis	9
Gangrene of genitals :—						
Penis	7
Scrotum	5
Laceration of genitals	3
Severe additional injuries :—						
Bladder	4
Rectum	4
Spine	5
Skull	1
Mention of impotence	10
Recorded deaths (mostly with fractured pelvis)	24

Care must be exercised in not looking upon the figures as a true percentage representation, but rather as an indication of the proportional value of any one complication. Thus 100 cases are actually admitted as having strictures and represent the minimum of such a complication. Of the remainder either their progress since discharge has not been given, or an overwhelming majority of those with adequate follow-ups have undergone repeated and regularized catheterizations. In fact the impression gained by this survey is that the shadow of stricture formation is a very ominous one, and it is hardly an exaggeration to state that the case of ruptured urethra that escapes long 'bougie' treatment must be the great exception rather than the rule. Kidd mentions that out of 26 follow-ups only one could be proved to be stricture-free, but later adds that of 25 complete ruptures 76 per cent were "practical" cures. Pasteau and Iselin mention only 3 out of 35 cases of urethral strictures as cured. The most tragic story of a strictured life is one of a patient who regularly catheterized himself every day for sixty-five years following a rupture, and finally succumbed to a carcinoma on that part of the bladder which had received a daily touch from the head of the instrument (Stirling).

Table VII.—ANALYSIS OF 100 CASES OF STRICTURE

	NO. OF CASES
<i>Site :—</i>	
Bulb	38
Membranous	44
Uncertain*	18
<i>Cause :—</i>	
Falls 'astride'	36
Falls 'on side' without pelvic fracture	2
Falls 'on side' with pelvic fracture	36
Blows	5
Instrumental (1), unstated (20)	21
<i>Treatment :—</i>	
Suprapubic drainage	36
Perineal section	71
End-to-end anastomosis	36
In-dwelling catheter	79
Perineal wound drainage	31
<i>Sepsis :—</i>	
Wound	34
Fistula	25
Abscess	7
<i>Extravasation :—</i>	
Hours from accident to operation—under 24	18
Hours from accident to operation—under 48	5

* This figure is higher than the 'unstated' cases in *Table III*, for it is often noted that "out of a total of, say, 6 cases (4 bulbs, 2 membranous) there were 3 strictures", and no indication is given as to which type is meant.

A little comfort, however, can be extracted from this general gloom in that the word stricture is loosely used in two respects. First, a few cases are dubbed 'stricture' when a catheter fails to be passed during the early convalescence, and hurried retrogressions of sounds are done. Secondly, a 'soft' stricture is an unfortunate title. In the author's series, *Case 3* showed a 'soft' stricture on uroscopic examination at the eighteenth month, but the largest catheter his penis could carry slipped through the posterior urethra with the greatest ease. Four years later, with no intervening dilatations, there is no suggestion that the 'soft' stricture has interfered with micturition or with the urethrogram. Urethroscopy to-day would doubtless show the soft diaphragm; it might tempt the anxious to advise a long course of bougie treatment and to record the case without qualification as a 'stricture'.

Ralph Thompson and Lowsley give excellent summaries showing why traumatic strictures are worse than gonococcal ones, Thompson also stressing the serious nature of rupture in children.

RECORDED TREATMENT

It is impossible here to present every type of treatment employed in the 381 cases.

A *general* analysis (*Table VIII*) discloses that the perineum was incised in no less than 248 cases, and the bladder was opened in only 181. In but a few of

these 181—notably Banks's cases—was the bladder definitely recorded as closed at the end of the operation, and so 181 represents the very maximum of urinary deviation, i.e., just under 50 per cent. The in-dwelling catheter was used 180 times. In 105 cases it was employed as a splint to ensure the patency of the urethra, for a temporary suprapubic tube drew off the urine; in the remaining 75 cases the catheter alone served the dual purpose of splint and drain.

Table VIII.—TREATMENT OF RUPTURED URETHRA

TREATMENT	NO. OF CASES
Perineal section	248
End-to-end anastomosis	141
In-dwelling urethral catheter :—Membranous (92); bulb (83); penile (2), female (3)	180
Perineal wound drainage	41
Suprapubic drainage	181
Perineal bladder drainage proximal to suture ^{51, 63, 71}	3
Urethral ends stitched to perineal skin	16
Ends too far apart to suture: Bridged by hinged urethral flap (3) ⁷⁰ ; bridged by fascia lata (2) ^{11, 51} ; bridged by saphenous vein (1) ⁵¹ ; left alone (6)	12
Use of silk or wax thread mandrin ^{34, 48, 52}	3

An *individual* analysis, however, justifies the following observations. There is almost universal agreement that bulbar and membranous ruptures should be differently treated. It is often stated that the deep situation and much less tendency to stricture formation of the membranous urethra submits more kindly to the in-dwelling catheter, yet it ought to be noted that the major portion of the 100 strictures implicated the membranous region. Bulbar injuries arouse argument as to the necessity for suprapubic deviation, the help or harm of an in-dwelling catheter, and whether or not an external urethrotomy should be made.

Marion emphasizes that immediate external urethrotomy (which he advocated in 1914) has no indication in traumatic rupture of the urethra. He prefers an instant suprapubic drainage as inaugurated by Rochet of Lyons. For membranous ruptures he relies on an in-dwelling catheter to maintain the axis of the channel, and in the large majority of such cases feels that perineal section is unnecessary. It should be reserved for the very few that develop strictures at a later date. In them a formal excision of scar tissue and end-to-end anastomosis can be performed, this time discarding the in-dweller on completing the suture. For bulbar injuries Marion dislikes anything more than simple deviation for six weeks. The perineum is now in a fit state for exploration and end-to-end suture over a resident sound. The urethra is rid of this sound at the end of the operation and convalescent bougie treatment is avoided. Marion is supported in his delayed operation by Coppidge, Raynaud, and others.

Young utilizes the in-dwelling catheter for both varieties, and reserves a suprapubic operation for retrogression, when desired, and for extravasation. He favours an immediate perineal incision, mid-line for the bulbar variety, and inverted V with

division of the central tendon and recto-urethralis muscle for the membranous lesions (*Figs. 220, 221*). Badly lacerated tissue is excised, and ends anastomosed or partial defects closed round a catheter which remains in situ for a week or more.

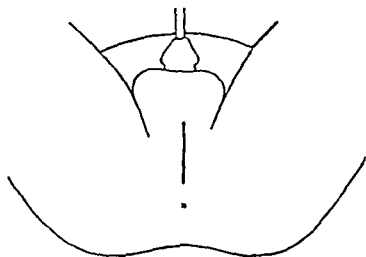


FIG. 220.—Young's incision for bulbar lesions.

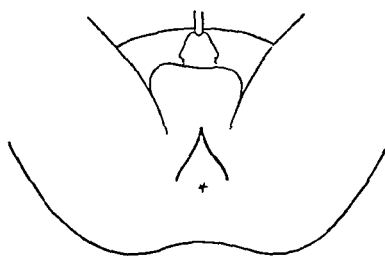


FIG. 221.—Young's incision for membranous lesions.

Young's method of repairing a membranous lesion is shown in *Figs. 222–223, A, B*. Where large urethral gaps occur it is only necessary to suture part of the mucous membrane over a large catheter.

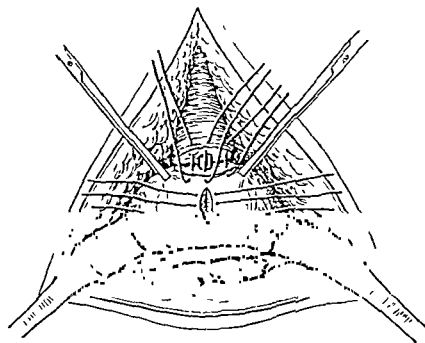
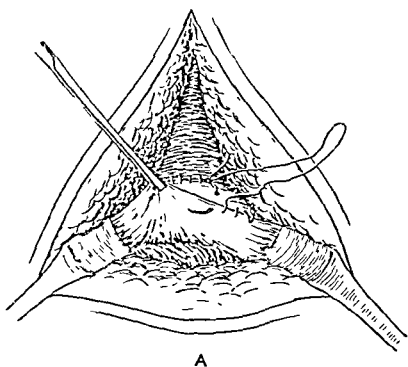
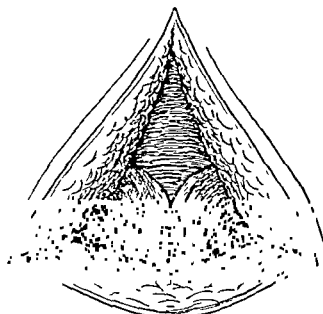


FIG. 222.—Young's method of repairing a membranous lesion. The radiating sutures do not pierce the mucous membrane. The small 'prostatic' incision draws off the urine, and allows a catheter to be passed down the urethra to find the damaged proximal end. An in-dwelling catheter remains in situ for seven days.



A



B

FIG. 223, A, B.—Young's method of closure of the deep wound in layers.

Legueu also operates at once, but condemns the in-dweller as a source of sepsis, and supports his contention by quoting experiments, in conjunction with Guyon, on dogs. His fear of stricture formation compels him to suture the bulbar

ends to the perineal skin. In the large majority these ends apparently sink back into their normal 'reconstructed' position, but in the few recalcitrant cases autoplasmic measures overcome the difficulty. Pasteau and Iselin support, and have popularized, Legueu's skin-suture method.

The *in-dwelling catheter* has been avoided by Alamartine, Cabot, Grey Turner, Garlock, Legueu, Pasteau, Iselin, and Rochet; but Banks, Becker, Smith, Mintz, and the majority of English and American writers have joined Young in favouring this catheter, not only for drawing off the urine, but also for maintaining the patency and splinting of the axis of the urethra during the healing stage.

In late septic cases, the reluctance to use the catheter *à demeure* appears to wane. Attwater maintained a catheter in such a type for four months with good effect, and his action has been endorsed by many of the French school.

Banks's simple but ingenious manœuvre may be used where the in-dweller seems to be of more accepted use, i.e., in membranous ruptures, and completely disposes of a perineal section. One metal sound with a cupped beak is retrograded through a tiny suprapubic incision. Another sound with a round tip is 'ante-graded' through the penis. A hand on each instrument carefully manipulates these together so that the rounded end fits into the cupped one. The sounds are then kept in firm apposition and gently swung round so that the penile instrument protrudes through the bladder wound. A soft rubber catheter is now tied to a small hole at its tip and pulled down the urethra as an in-dweller. The bladder is then closed.

Difficulty in replacing in-dwelling catheters has led some authors to pass a filiform bougie up the lumen of the old catheter before withdrawing the latter. A new rubber one has then been easily threaded over the central guide. McWhorter has employed with effect a wax silk mandrin. The loose ends hang from the penis and bladder and the suprapubic wound rapidly granulates around the latter. In one case this tiny seton remained threaded through the in-dweller up to the 5th day, and facilitated the guidance of dilating sounds along its path until the 65th day. The 'railroad' method of replacing catheters is too well known to be described here; it suffers the disadvantage of having to be catered for at the original operation.

Failure to pass a bougie has been variously circumvented. Marion is prepared philosophically to wait a few more days until the reactionary œdema has settled down rather than attempt to pass a catheter a second time at one sitting. A curved Béniqué after the Buckston Browne style will now often negotiate the slight angulation of a urethral axis which a straight catheter cannot explore. The gentle 'clocking of the compass' by one filiform; the simultaneous passing of several filiforms, each one in turn being coaxed a little further down the urethra until one eventually finds the proximal channel; and special urethroscopic instruments for passing a sound under direct vision (Lowsley, Raynaud),—have all been used to overcome the difficulty.

The few *flaps* that have been fashioned to bridge gaps indicate their extremely rare and doubtful application. One inventive method is given (Fig. 224), but a perfection of the author's weight extension method avoids the necessity for cutting into the normal urethra to make the flaps.

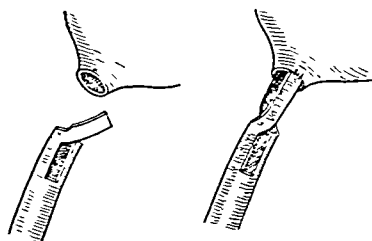


FIG. 224.—Watson's method of swinging round two lateral urethral flaps to bridge a gap.

The penile injuries have presented no difficulty except that a 'nasal' cautery was used to sear the site of one furious 'coital' bleeding.

Extravasation when it occurs must be given priority over all attempts at repair. Hartman's and Marion's methods of dealing with it are shown in *Figs. 225, 226, A, B.*

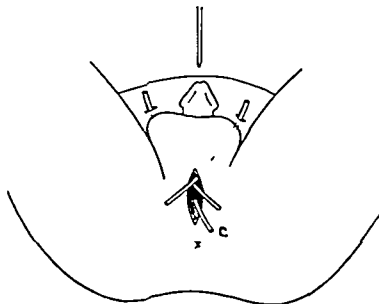
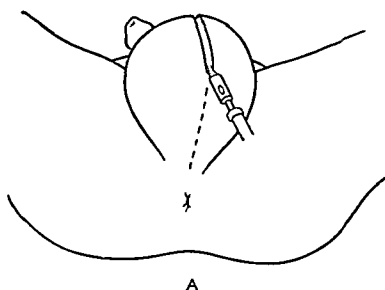
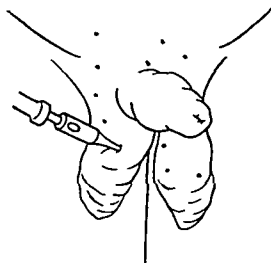


FIG. 225—Hartman's method of dealing with severe extravasation of urine. The bladder is drained from the perineum by the catheter, C.



A



B

FIG 226, A, B—Marion's cautery method of dealing with severe extravasation of urine.

DISCUSSION

Successful anastomosis in the colon is so dependent on a good blood-supply that moderate sepsis or filthy bowel contents seem to be of lesser account. It might well be supposed that the rich vascularity of the other excretory channel would even more ensure recovery of extensive urethral injury or infection, and it is surprising to find strictures so common. An inherent tendency to scar-tissue formation or a damage to nerves—impotence and atrophy figuring so little—are causes which can be dismissed almost as certainly as the circulatory one. *Sepsis seems to be the one tangible cause which is capable of control.*

Causes of Sepsis.—Sepsis may arise from the following: (a) The urine; (b) The in-dwelling catheter; (c) Haphazard ascending catheterization; (d) Proximity of perineal wound to anus; (e) The extensive bruising and hæmatoma formation; (f) Concomitant injuries.

The Urine.—When urine repeatedly passes over a raw area it invites the septic necrosis familiar in extravasation. Protection must therefore occur both before and after surgical intervention.

Ambulance men and house surgeons will do well to warn patients not to pass water, and specimens should not be secured just to see whether bladders or urethræ are intact.

At operation, the most certain way of abstracting the urine should be employed. Thomson-Walker has shown that urine leaks along an evacuating catheter, so it seems logical to do a suprapubic deviation in all cases, partial or complete. Injuries of the pendulous urethra are the only possible exceptions. Marion deserves credit for emphasizing the need for suprapubic drainage, and Hugh Cabot condensed the subject best to the author in 1931 by saying—"when operating on any waterworks you must divert the stream".

If any reason prevents immediate admission to hospital, repeated suprapubic aspiration through a long needle is an excellent step. The case of a sailor whose injury occurred five days from port, and whose bladder was regularly tapped by the ship's surgeon, cannot be quoted too often, for not the slightest sign of extravasation could be found at the major operation ashore.

The In-dwelling Catheter.—The in-dwelling catheter as a source of sepsis has been condemned on the grounds that even the normal urethra becomes septic when a catheter is tied in for three to four days. In its condemnation Grey-Turner expresses the view-point best by stating that no foreign body, such as a catheter, must come into contact with the urethral wound during healing. The present paper suggests that as no less than 80 per cent of the 100 strictures (*Table VII*) harboured an in-dweller, the instrument may indeed be a cause of stricture rather than a cure.

Haphazard Anterior Catheterization.—Organisms can always be grown from the normal anterior urethra, and careless 'sounding' will carry these up to any raw area; furthermore such sounding may inadvertently increase the damage. Catheterization must only be undertaken in the operating theatre where major surgery can immediately follow if necessary. In fact it is advisable to avoid ascending catheterization as a diagnostic measure. It is felt that a good history and physical signs are sufficient to justify a suprapubic cystotomy; whether the rupture is partial or complete is better established by gentle retrogression of a catheter down the urethra. Should the tip succeed in appearing at the penile meatus, the funnel end should be cut off and the catheter pulled downward and discarded, rather than be drawn up to risk meatal contamination. The abdominal incision has the added advantage of immediately allaying doubt about a ruptured bladder or extravasation.

One could only explain why the old man (*Case 1*) made such a splendid recovery and the healthier young man (*Case 2*) had such a septic course on some such grounds as this. The one difference in the treatment of these two *partial* ruptures—both operated upon within an hour of the accident, and both even subjected to an immediate suprapubic urinary diversion—was in their catheterization. In *Case 1* retrogression alone was employed; in *Case 2* 'antegression'. It is unlikely that the one preliminary voiding of 'sterile' urine in *Case 2* fouled the track, and so the ascending catheter must be suspected as the vehicle of infection.

Proximity of Perineal Wound to Anus.—The perineal skin is a poor site, especially when bruised, for aseptic exploration or external urethrotomy.

Extensive Bruising and Hæmatoma Formation.—These sources of sepsis are generally admitted. The opening of the perineum, removing pulped tissue and blood-clot, and leaving in a small drain to evacuate the oozing of the first twenty-four hours, therefore might be considered an advantage, but the dangers of increasing the sepsis by anal contamination, of excising too much damaged tissue on account of the impossibility of knowing how much is going to recover, and of

finding a poor foothold for exact end-to-end sutures, all suggest that operation may well be postponed till adequate resolution and absorption of blood-clot has occurred. Large perineal hæmatomata alone require evacuation at the preliminary derivation.

Delay in Treatment.—So great are the dangers of infection and extravasation that a ruptured urethra should be considered as much an emergency as a ruptured peptic ulcer. An adequate blow or fall with penile bleeding, or even repeated desire to micturate, should be sent to hospital for urgent attention and possible diversion of the urinary stream.

Concomitant Injuries.—Ruptures of the rectum have been recorded by Berry, Haines, Jastrum (Case 12), Young, André (female). Where these patients have recovered from such grosser damage—in one, the abdominal aorta could be seen from the perineum—the rectal wound has healed very promptly but the urethra has occasioned late anxiety.

CONCLUSION

Excluding the exceptional lacerations just described, and whose severity makes them purulent from the beginning, careful attention to detail will do much to reduce the toll of sepsis.

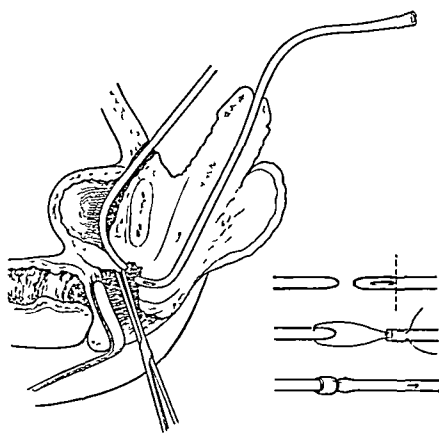


FIG. 227.—Showing a side view of the perineal approach to the membranous urethra, and the method of joining the catheters to enable one to be pulled down the urethra to act as an in-dweller.

In All Cases.—Success will depend on :—

Admission to hospital as soon as possible after the accident and before micturition has been allowed.

A careful toilet of any proposed operative site, particularly of the perineum, penis, and anterior urethra.

The prompt diversion of the urinary stream by a suprapubic incision, if necessary under a local anæsthetic.

The retrogression of a 'boiled' gum-elastic catheter down the urethra to establish a diagnosis of partial or complete rupture, and the discarding of this catheter from the meatal rather than the bladder end.

The wrapping of the penis in a sterile dressing which cannot fall off, to prevent ascending infection during the healing stage.

The high blocking of the foot of the bed (allowing several pillows under the head for comfort), to ensure the urine flowing away from the meatus into the fundus of the bladder.

The attachment of a Sprengel's pump to the suprapubic tube.

In Membranous Ruptures.—The disquieting figures of *Table VII* reveal that stricture is just as common in the membranous urethra as in the bulb, and as an in-dwelling catheter has formed the major treatment, almost 80 per cent of these cases, it would seem fair to question its propriety and to suggest it may well be replaced by a simple instant suprapubic drainage and a formal late repair. If an in-dweller is still preferred, it should be inserted, where possible, by retrogression, without resorting to a perineal incision to guide the catheter across the ruptured site.

Should this be difficult, an ascending catheter, after suitable lavage, and according to the method of Banks, will assist in straightening out the distal end for the reception of the descending catheter.

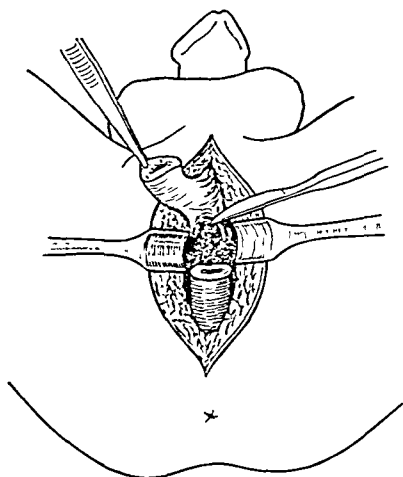


FIG. 228.—Marion's method of mobilizing the distal urethra to overcome the gap left after excision of the traumatized area.

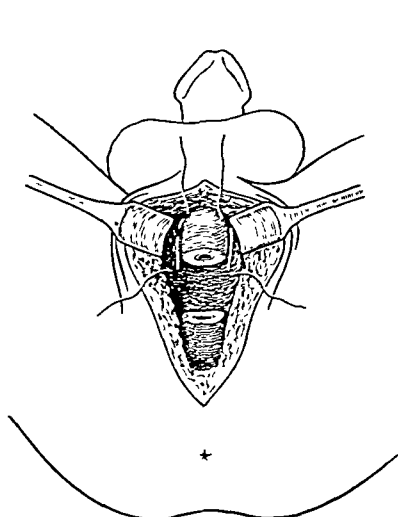


FIG. 229.—Marion's stay-sutures, when tied, draw the distal end up to the proximal one. Additional long mattress sutures can be inserted on the outside of the urethra to relieve the tension on the small radial sutures (Fig. 230), much as in the repair of a cut tendon.

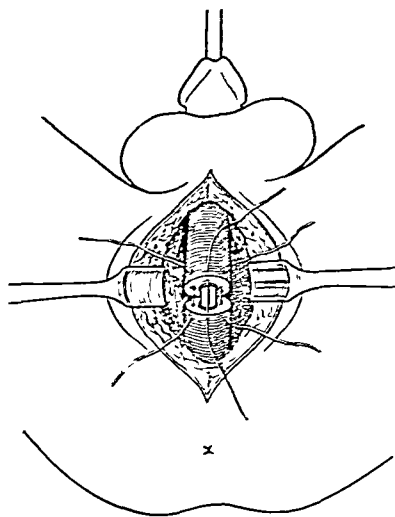


FIG. 230.—Marion's stay-sutures tied, the ends approximated and the small radial sutures inserted so that they do not pierce the mucous membrane. The in-dwelling catheter is discarded at the end of the operation.

The author's method of avoiding primary perineal incision by seizing the ascending catheter from the depth of the suprapubic wound and joining it to the retrograded one may be used when there is a dislocation of the prostate or separation of the symphysis pubis (Cases 3 and 4). The pulling down of the bladder and thus eliminating any gap between the torn ends, by a spring apparatus (similar

to a prostate bag-spring) or weight extension (as devised for *Case 4*), is a logical attempt at avoiding further surgery. Such a method is simple and extraordinarily well tolerated by the patient, but has only been used once, and is not free from the assumed disadvantage of an in-dweller.

When the perineal approach is used, the necessary steps of the operation and the method of uniting the catheters are shown in *Fig. 227* and *Figs. 221–223 B*.

In Bulbar Ruptures.—The in-dwelling catheter is discarded and the best means of diverting the urine (a suprapubic) is essential for a varying period up to six weeks. A more certain union of the torn fragments is then possible. The freshened ends are united by small radial sutures to prevent angulation or mal-alignment, and tension is removed from these binding sutures by long mattress stitches placed on the outside of the corpus spongiosum, much as in the repair of a cut tendon. Marion's manoeuvre (*Figs. 228–230*) should be employed to mobilize and fix the distal end if there is any doubt about tension or the traversing of gaps.

The anastomosis completed, the deep wound is closed in two or three layers, and a small drain inserted for twenty-four hours at the posterior end of the skin wound to control any temporary exudation. The patient is nursed with his legs wide apart (contrast the healing of an axillary wound with the arm abducted and to the side), and the bowels are constipated for five days. Each motion induces another toilet of the wound and change of dressings.

There is no urethral catheterization for 14 to 21 days, and on the first occasion it is accompanied by an anterior urethral wash-out under local or general anaesthesia to allay pain and spasm.

The suprapubic wound should only be allowed to close when the urethra is completely healed and free from catheter difficulty.

Any such difficulty is best dealt with by direct urethroscopy. Obstruction such as a small scar-tissue sail can be treated, or the proximal end can be seen to allow the gentle introduction of the first bougie of serial dilators. The watch-word '*doucement*' is at all times appropriate.

In Penile Ruptures.—Penile ruptures are happily rare, and except in extensive lacerations respond readily to simple means.

In Female Ruptures.—These must be just as carefully handled as bulbar injuries in the male. In-dwelling catheters are not well tolerated, and suprapubic cystostomies are preferred as soon as possible.

SUMMARY

1. Details are given of 8 personal cases and 381 recorded cases of traumatic rupture of the urethra.

2. The diagnosis is nearly always easy, and can be obtained from the history of an 'adequate' accident and from the simple physical signs. Passing a catheter is a most unreliable means.

3. Difficulty is only experienced in grosser injuries to the pelvis, spine, or rectum, for here the bladder or urethra may also be involved.

4. The physical signs are shock, pain, bleeding from the penis, perineal hæmatoma, ecchymosis, and abnormality of micturition.

5. Stricture formation is more common and serious after traumatic ruptures than after any other type. In this series stricture appears to be as common after membranous as after bulbar injuries.

6. Sepsis is suggested as the one cause of stricture formation which can be controlled.

7. The various methods of repair are summarized.

8. Treatment is urged as soon as possible after the accident, and is directed against fouling of the raw area by urine, haphazard catheterization, or perineal contamination.

9. Suprapubic deviation is submitted as essential in all ruptures, partial or complete. The only exception to this rule is the rare injury to the penile shaft.

10. Posterior or 'membranous' ruptures are said to tolerate an in-dwelling catheter well and only a few cases produce strictures or require perineal exploration. The series does not endorse this general belief. Immediate perineal incisions are best avoided. A new emergency method of approximating the ends is described. Future treatment may more nearly approach that of injuries to the bulb.

11. Bulbar ruptures are certainly prone to stricture formation. In such lesions a suprapubic cystotomy is instantly performed, but a formal external urethrotomy is postponed until bruised tissue has recovered, necrotic tissue is well differentiated, and accurate end-to-end suture approximation has a better chance of 'holding' and healing.

12. Penile injuries are of less serious moment, and may not require a supra-pubic operation.

13. Female injuries require immediate diversion of the urinary stream.

It is a pleasure to record my thanks to the late Mr. R. P. Rowlands for giving me *Cases 1, 2, 3, and 5*, and for urging me to publish the emergency measures used in *Case 3*; to Mr. Davies-Colley for allowing me to use a similar method on *Case 4*; to Mr. E. C. Hughes for *Case 6* and Mr. O. L. Addison for *Case 8*. I am indebted to Dr. H. M. Worth for urethrograms of cases 1, 2, 4, and 5, to Dr. Bertram Shires for the urethrogram of case 3, to the Library staff of the Royal Society of Medicine for supplying me with books, to Mr. Steward of the Royal College of Surgeons for his beautiful diagrams, and to Miss Davies-Colley for the photograph.

REFERENCES

(The number of cases taken are shown in brackets at the end of the reference.)

- ¹ ALAMARTINE, M., *Lyon Chir.*, 1922, xix, 82. [1.]
- ² ALLISON, J. R., *Mil. Surgeon*, 1920, xlvii, 324. [1.]
- ³ ANDRÉ, *Bull. Soc. d'Obst.*, 1932, xxi, 286. [1.]
- ⁴ ATTWATER, H. L., and PREISKEL, I., *Lancet*, 1933, ii, 1424. [1.]
- ⁵ BAILEY and HUDDY, *Brit. Jour. Surg.*, 1925, xii, 183. [Nil.]
- ⁶ BANKS, H., *Ibid.*, 1927, xv, 262. [3.]
- ⁷ BARNETT, C. E., *Urol. and Cutan. Rev.*, 1920, xxiv, 692. [2.]
- ⁸ BECKER, J., *Arch. f. klin. Chir.*, 1934-35, clxxxi, 317. [20.]
- ⁹ BERRY, N. E., *Canada Med. Assoc. Jour.*, 1930, xxii, 475. [32.]
- ¹⁰ BESLEY, F. A., *Surg., Gynecol. and Obst.*, 1927, xliv, 372. [Nil.]
- ¹¹ BRENNER, E. C., *Jour. of Urol.*, 1924, xi, 197. [1.]
- ¹² CABOT, HUGH, Personal communication. [Nil.]
- ¹³ CARRARO, D. N., *L'Osp. maggiore*, 1918, vi, 155. [1.]
- ¹⁴ CLIFFORD-ABBOTT, A., *Canada Med. Assoc. Jour.*, 1929, xx, 634. [1.]
- ¹⁵ COMBES, W. L., *U.S. Naval Med. Bull.*, 1921, xv, 407. [2.]
- ¹⁶ COPPRIDGE, W. M., *South. Med. Jour.*, 1935, xxviii, 69. [8.]
- ¹⁷ CUMSTON, C. G., *N.Y. Med. Jour.*, 1914, xcix, 10. [Nil.]
- ¹⁸ DAVIS, R. T., *Surg. Gynecol. and Obst.*, 1927, xlv, 836. [1.]

- ¹⁹ DEAN-BEVAN, A., *Surg. Clin. Chicago*, 1918, ii, 1131. [1.]
- ²⁰ DELZELL and STEVENS, *Jour. of Urol.*, 1935, xxxiv, 372. [35.]
- ²¹ DUBOUCHER, M., *Bull. et Mém. Soc. nat. de Chir.*, 1926, lii, 1214. [1.]
- ²² DUROSELLE, M., *Bull. et Mém. Soc. Chir.*, 1927, lviii, 142. [1.]
- ²³ EDMUNDS, A., *Med. Press and Circ.*, 1913, xcvi, 558. [1.]
- ²⁴ FIGUEIRA, M., *Long I. Med. Jour.*, 1918, xii, 194. [2.]
- ²⁵ FLANDRIN, P. R., *Jour. d'Urol.*, 1929, xxvii, 439. [3.]
- ²⁶ FLECHTENMACHER, C., *Zeits. f. Urol.*, 1921, xv, 73. [1.]
- ²⁷ FOULDS, G. S., *Canada Med. Assoc. Jour.*, 1930, xx, 365. [2.]
- ²⁸ FRANKENTHAL, L., *Zentralb. f. Chir.*, 1925, lii, 1598. [1.]
- ²⁹ FRUCHAUD, H., *Jour. d'Urol.*, 1934, xxxviii, 167. [1.]
- ³⁰ GARLOCK, J. G., *Surg. Gynecol. and Obst.*, 1923, xxxvii, 533. [1.]
- ³¹ GREY-TURNER, G., *Lancet*, 1923, ii, 82. [Nil.]
- ³² GRIMAUDT, M. L., *Bull. et Mém. Soc. Chir.*, 1926, lii, 1232. [4.]
- ³³ HAINES, C., *Jour. of Urol.*, 1933, xxix, 285. [3.]
- ³⁴ HEITZ-BOYER, *Jour. d'Urol.*, 1935, xl, 438. [1.] *Bull. et Mém. Soc. Chir.*, 1921, xlvii, 584. [1.]
- ³⁵ HENRY, M. R., *Paris chir.*, 1920, xii, 267. [1.]
- ³⁶ HOLLANDER, H., *Berlin klin. Woch.*, 1918, lv, 219. [1.]
- ³⁷ HOPKINSON, H. C., *Lancet*, 1935, ii, 428. [1.]
- ³⁸ HUG, M. E., *Jour. d'Urol.*, 1934, xxxviii, 551. [3.]
- ³⁹ HUTTER, K., *Zeits. f. urol. Chir.*, 1935, xli, 260. [1.]
- ⁴⁰ JANET, J., *Jour. de Méd.-Chir. prat.*, 1935, cvi, 305. [3.]
- ⁴¹ JASTRAM, M., *Deut. Zeits. f. Chir.*, 1923, clxxvii, 70. [12.]
- ⁴² KESSELER, J., *Jour. d'Urol.*, 1932, xxxiv, 45. [1.]
- ⁴³ KIDD, F., *First Con. Soc. Inter. d'Urol.*, 1921, i, 169. [41.]
- ⁴⁴ LEGUEU, M. F., *Bull. et Mém. Soc. Chir.*, 1927, liii, 92. [Nil.] *Bull. et Mém. Soc. nat. de Chir.*, 1927, liii, 92. [47.]
- ⁴⁵ LOWSLEY, O. S., *Urol. and Cutan. Rev.*, 1935, Nov., 776. [Nil.]
- ⁴⁶ LUCCHESI, G., *Polislinico (Sez. prat)*, 1934, xli, 1610. [1.]
- ⁴⁷ MANEN, S. A., *Annales de Pédiat.*, 1934, vii, 442. [1.]
- ⁴⁸ MCWORTER, G. L., *Surg. Gynecol. and Obst.*, 1927, xlii, 247. [4.]
- ⁴⁹ MARION, G., *Traité d'Urologie*, 1935, ii. [Nil.] *Bull. et Mém. Soc. Chir.*, 1927, liii, 9. [Nil.] *Jour. d'Urol.*, 1914, v, 553. [10.]
- ⁵⁰ METRE, P. W. VAN, *China Med. Jour.*, 1916, xxx, 335. [1.]
- ⁵¹ MORISON, R., *Surgical Contributions*, 1916, i, 271. Bristol: Wright. [2.]
- ⁵² MURARD, M., *Lyon méd.*, 1921, cxxx, 738. [2.]
- ⁵³ PAPIN, M. F., *Bull. et Mém. Soc. méd. Bordeaux*, 1918. [1.]
- ⁵⁴ PASTEAU and ISELIN, *First Con. Soc. Inter. d'Urol.*, 1921, 124. [19.]
- ⁵⁵ PARTSCH, F., and BREITLANDER, *Zeits. f. urol. Chir.*, 1928, xxv, 108. [2.]
- ⁵⁶ PEACOCK and HAINES, *Jour. of Urol.*, 1926, i, 563. [1.]
- ⁵⁷ PLANZ, J., *Beitr. z. klin. Chir.*, 1934, clxx, 111. [13.]
- ⁵⁸ POWERS, J. H., and SMITH, N.Y. *State Jour. of Med.*, 1932, xxxii, 1188. [2.]
- ⁵⁹ REYNARD, M., *Lyon méd.*, 1928, cxlii, 41. [1.]
- ⁶⁰ ROUSSEAU, M. F., *Paris chir.*, 1921, xiii, 233. [1.]
- ⁶¹ SESHACHALAM, T., *Ind. Med. Gaz.*, 1932, lxvii, 85. [1.]
- ⁶² SMITH, F. W., *Amer. Jour. Surg.*, 1916, xxx, 394. [1.]
- ⁶³ SMITH, G. G., and MINTZ, E. R., *Trans. Amer. Assoc. Gen.-Urin. Surg.*, 1931, xxiv, 241. [30.]
- ⁶⁴ SPURR, R., and MONTANARE, H., *Rev. d. Especialid.*, 1930, v, 117. [1.]
- ⁶⁵ STARLINGER, F., *Wien. klin. Woch.*, 1926, xxxix, 1515. [1.]
- ⁶⁶ STIRLING, W. C., *Jour. of Urol.*, 1927, xvii, 503. [7.]
- ⁶⁷ TANNER, E. K., *Long I. Med. Jour.*, 1918, xii, 273. [2.]
- ⁶⁸ THOMPSON, A. R., *Brit. Jour. Child. Dis.*, 1920, xvii, 70. [4.]
- ⁶⁹ TURLEY, H. K., *Urol. and Cutan. Rev.*, 1934, xxxviii, 772. [13.]
- ⁷⁰ WATSON, E. M., *Jour. of Urol.*, 1935, xxxiii, 64. [3.]
- ⁷¹ WHEELER, Sir W. I. DE C., *Brit. Jour. Urol.*, 1929, i, 126. [7.] *Proc. Roy. Soc. Med.*, 1928, xxii, 1.
- ⁷² YOUNG, H. H., *Practice of Urology*, 1926, ii, 595. [Nil.] *Jour. of Urol.*, 1929, xxi, 417. [Nil.] *Zeits. f. Urol.*, 1929, xxiii, 419. [9.]

HYPERPLASTIC TUBERCULOSIS OF THE STOMACH CAUSING HOUR-GLASS DEFORMITY, WITH COMPLETE SQUAMOUS METAPLASIA OF THE UPPER LOCULUS

BY G. W. WATSON, E. R. FLINT, AND M. J. STEWART, LEEDS

HYPERPLASTIC tuberculosis of the digestive tract occurs most frequently in the region of the lower ileum, cæcum, and ascending colon, where it usually takes the form of a palpable mass with symptoms of chronic intestinal obstruction. Rarely, a similar form of the disease affects the pyloric portion of the stomach, again with the formation of a tumour and slowly progressive obstruction. In both sites, but more especially in the stomach, the clinical diagnosis is likely to be one of carcinoma.

The case which we now report is remarkable in that the granulomatous lesion has affected almost exactly the middle of the stomach, producing a high-grade hour-glass constriction, and there is the additional peculiarity that the mucosa of the upper loculus has been almost completely replaced by squamous epithelium. In a careful search of the literature we have failed to find any record of a similar case.

CASE REPORT

CLINICAL HISTORY.—F. S., a male, aged 31, was admitted to a medical ward of the General Infirmary at Leeds on August 21, 1935, with a three-months' history of epigastric pain, vomiting, and loss of weight. He was a tailor's presser, married, and had been deaf and dumb from birth. Vomiting of *undigested food* occurred about an hour after a meal and this afforded some relief to the pain, which was aggravated by the ingestion of more food. Both pain and vomiting became progressively more severe, and at the time of admission only small quantities of fluid could be retained. He had lost over a stone in weight. Prior to May, 1935, he had had no illnesses, and there was no family history of tuberculosis. He had always been a man of moderate habits.

ON EXAMINATION.—During his stay in the medical ward he was thoroughly investigated, the salient points being as follows: He was emaciated and dehydrated. Physical examination revealed little beyond epigastric tenderness; no tumour could be felt in the abdomen, and there was no visible peristalsis. The bowels had been constipated only since the beginning of the illness. Micturition was normal and the urine contained no abnormal constituents. The Wassermann reaction was negative. On August 29 a fractional test-meal showed complete achlorhydria, with low total acidity and rapid emptying of the stomach. A blood-count on September 12 showed red blood-corpuscles



FIG. 231.—X-ray photograph taken on October 1, 1935, almost immediately after the administration of a second test-meal, the first having been given six hours previously. It shows a constriction of the middle portion of the stomach, with a large quantity of barium in the upper loculus and only a very small amount in the lower.

4,700,000, Hb 84 per cent, colour index 0.9, white blood-corpuscles 6,800. There were no abnormal features in stained films. X-ray examination on October 1 revealed a small narrow stomach with a well-marked stricture in the middle of the organ (*Fig. 231*). A small quantity of the barium meal was held up for some time at the cardiac end, and it was found impossible to fill the pyloric portion. The screen appearances suggested neoplasm. The œsophagus was normal.

On a diagnosis of carcinoma of the stomach, probably of leather-bottle type, the patient was transferred to a surgical ward for operation and was put on continuous intravenous glucose-saline for several days.

OPERATION.—This was performed on October 11 under spinal anaesthesia. The stomach presented itself as a short firm organ some six inches in length and not more than an inch across. There was a hard mass occupying the middle portion, and the whole organ was tucked up underneath the ribs. The cardiac end appeared fairly normal, except that it looked as though it would hold only about as much as a small coffee cup. The appearances as a whole were suggestive of leather-bottle stomach, yet the wall did not seem to be permeated

in the manner characteristic of this condition, and doubt was felt and expressed as to the true nature of the lesion. There were no palpable glands. As the history and the radiological and chemical findings all supported the diagnosis of cancer, and since practically no useful part of the stomach was free from disease, it was decided to remove the whole organ. Even if it proved to be malignant there seemed to be a reasonable prospect that the whole of the disease might be eradicated. Total gastrectomy was accordingly performed and the œsophagus anastomosed to the jejunum, a loop of which was brought through the transverse mesocolon. The usual two layers of sutures were employed.

AFTER-HISTORY.—The patient stood the operation well and made an uneventful recovery. Continuous intravenous glucose-saline was kept going for two days, until it was felt safe to allow fluids in sufficient quantity by the mouth. On October 24, thirteen days after operation, a blood-count showed red blood-corpuscles 4,100,000, Hb 80 per cent, colour index 0.9, white blood-corpuscles 7,500, with normal films. When discharged from hospital on November 20 he was making steady progress towards recovery.

The patient was re-examined on February 3, 1936. He was eating well, without restriction as to quantity or kind of food, and had gained half-a-stone in weight. A blood-count showed red blood-corpuscles 5,100,000, Hb 90 per cent, colour index 0.9, white blood-corpuscles 6,800, with normal films. Radiological examination showed no delay in the passage of the barium meal, which reached the cæcum in four hours. There was no appreciable dilatation of the small intestine near the anastomosis (*Fig. 232*).

DESCRIPTION OF THE GROSS SPECIMEN.—The stomach, the whole of which has been excised, is very greatly reduced in size, measuring no more than 4 in. along the lesser curvature and about 7 in. along the greater curvature. There is a notable constriction involving fully an inch of the middle of the organ, producing a well-marked hour-glass deformity. There is, however, no evidence of scarring on the serosal aspect nor of involvement of the peritoneum by new growth. When the organ had been laid open along the line of the greater curvature (*Fig. 233*) the central constriction was found to be due to a lesion of granulomatous aspect, causing the whole of the viscus to be much thickened (up to half an inch), leaving a lumen no



FIG. 232.—X-ray photograph taken on February 3, 1936, four months after total gastrectomy and immediately after the administration of a second test-meal. There is flooding of the loops of jejunum with barium, and no evidence of holding up in the œsophagus. The whole of the test-meal given six hours previously is collected in the cæcum and ascending colon.

larger than a small quill. The lower loculus presented for the most part a normal-looking mucosa, except that the lesser curvature region for fully one inch below the constriction was the seat of ulceration of peculiar type. The margin of the ulcer was sinuous, red, and slightly undermined, the floor nodular and partly covered by a thin necrotic layer. The pyloric ring and adjacent mucosa appeared normal. The upper loculus presented an entirely different appearance. It had a smooth, white, firm lining at once suggesting squamous epithelium, except that along the line of the greater curvature there was a slightly projecting band of what appeared to be bright-red granulation tissue extending upwards for fully two inches. No gastro-oesophageal junction could be made out. In contradistinction to the lower loculus, the wall here showed great fibrous thickening of the submucosa. This appeared to be two to three times the thickness of the muscular coat, from which by reason of its whiteness it was very clearly demarcated, the muscularis being reddish in tint. The granulomatous lesion causing the hour-glass constriction was provisionally regarded as either tuberculous or syphilitic.

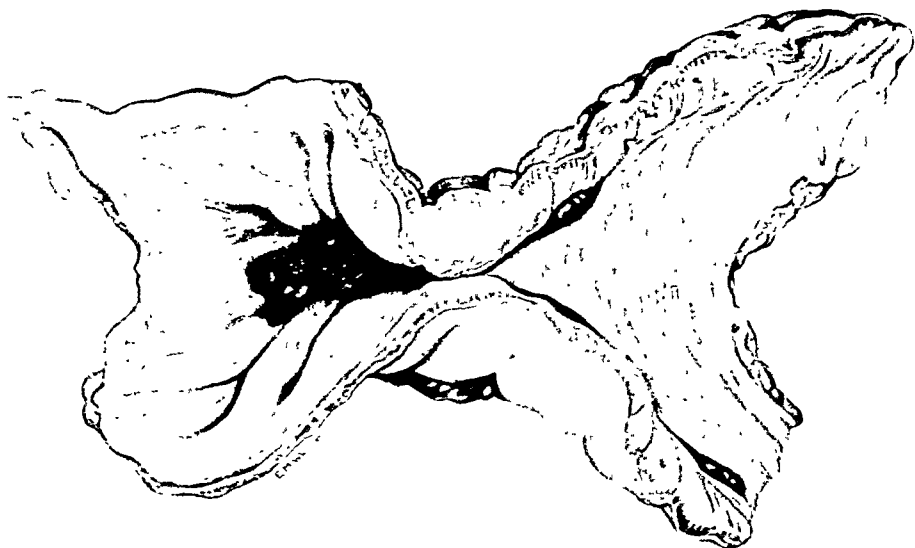


FIG. 233.—Stomach laid open along the greater curvature, showing the granulomatous and ulcerated lesion of the middle portion and the squamous-epithelial-lined upper loculus. Note the great fibrous thickening of the submucosa in this region. ($\times \frac{1}{2}$.)

HISTOLOGY.—

1. The granulomatous lesion in the centre of the organ presents the characters of hyperplastic tuberculosis. The lesion for the most part is devoid of tubercle follicles or giant cells, the ulcerated surface being clothed with a broad zone of highly vascular non-specific granulation tissue. Beneath this there is a greatly fibrosed and thickened submucosa which sends numerous strands of fibrous tissue into the underlying muscular coat. Here and there, however, both in the midst of the granulation tissue and deep in the fibrous zone, there are scattered, somewhat atypical, tubercle follicles, often perivascular in distribution, some with, some without, characteristic Langhans-type giant cells with peripherally arranged nuclei (Fig. 234). Tubercle bacilli have not been demonstrated in the follicles. A section of the ulcerated area extending along the lesser curvature of the lower loculus shows only very occasional ill-formed tubercle follicles. For the most part the lesion here consists of a narrow zone of non-specific, highly vascular, granulation tissue implanted directly upon the muscular coat (Fig. 235). The lower margin of the ulcer is undermined, the angle being occupied by a mixture of necrotic material and pus.



FIG. 234.—A tubercle follicle, with central area of epithelioid cells and marginal zone of lymphocytes in relation to which lie two giant cells of Langhans type.



FIG. 235.—Floor of lower ulcerated portion of the mid-gastric lesion, showing (above) a thick layer of highly vascular, mainly non-specific, granulation tissue, and (below) the somewhat fibrosed muscular coat.



FIG. 237.—Another portion of the upper loculus, showing squamous epithelial lining with broad stratum lucidum. Submucosa thickened and fibrosed. The section does not extend as deep as the muscular coat.



FIG. 236.—Section of upper loculus, showing squamous epithelial lining, hypertrophied muscularis mucosae, thickened and fibrosed submucosa, and hypertrophied muscular coat.

2. Sections taken from various positions in the upper loculus show that it is everywhere lined by stratified squamous epithelium (*Figs. 236-238*). The epithelium is many layers thick and shows in places a well-marked stratum lucidum (*Figs. 237, 238*), but there is no actual keratinization. Nuclei are present even in the most superficial layers. In one place only (*Fig. 238*) in three blocks examined is there a group of mucus-secreting glands of gastric type devoid of oxyntic cells. Everywhere there is a very well-developed muscularis mucosæ, several times the normal thickness, with varying amounts of fibrous tissue lying amongst the unstriped-muscle bundles (*Figs. 236, 237*). The submucosa is much fibrosed and in places is as much as 5 mm. in thickness. Small aggregations of lymphocytes are present in the more superficial layers, but for the most part the submucosa consists of very densely collagenous, comparatively acellular, fibrous tissue.



FIG. 238.—A third portion of the upper loculus, showing a few gastric glands lying beneath the squamous lining. The rest of the section shows only muscularis mucosæ and submucosa.

3. The muscular coat consists of two main layers, a broad inner and a narrow outer (*Fig. 236*), with well-developed Meissner's plexus between. The inner part of the inner coat is much broken up in places by strands of fibrous tissue, especially in relation to the granulomatous lesion, but also in the walls of both the upper and lower loculus. In the lower loculus, in the neighbourhood of the granulomatous lesion, the muscle bundles are separated by loose, œdematous, and highly vascular tissue with many inflammatory cells.

4. The mucosa of the lower loculus is thick and well developed, with a thick muscularis mucosæ much overrun by lymphocytes and plasma cells. There is a certain amount of chronic gastritic change, with excess of lymphoid tissue in the mucosa.

COMMENTARY

Pathological.—This case presents a number of interesting and unusual features :—

1. Hour-glass stomach is nearly always due to cicatricial contraction in the floor of a large chronic peptic ulcer lying astride the lesser curvature. Occasionally a sclerosing carcinoma of slow evolution may produce a somewhat similar type of deformity. In the present instance the lesion is granulomatous, having the characters, in our opinion, of hyperplastic tuberculosis.

2. Tuberculosis of the stomach in any form is generally regarded as rare. From time to time the routine histological investigation of this organ reveals the presence of miliary tubercles in the mucosa or submucosa, often in association with other lesions, peptic or neoplastic (Collinson and Stewart, 1927-28). Tuberculous ulcers, when they occur, are usually part of a widespread ulcerative process throughout the digestive tube, and, like the miliary lesion, are of little clinical interest or importance. Occasionally, however, they are due to tuberculous glands or peritoneal nodules ulcerating through the gastric wall. Hyperplastic tuberculosis is the rarest form of all. As in the corresponding ileo-cæcal lesion, it takes the form of a circumscribed tumour-like mass which tends to encircle the gut, and, as its chief site of occurrence is the pyloric portion of the organ, it leads presently to obstructive phenomena which are likely to be attributed to neoplasm. If, as is usual, a palpable tumour is present in the epigastrium, or if a test-meal shows achlorhydria, the diagnosis of gastric carcinoma can hardly fail to be made. The occurrence of a similar type of lesion in the mid-gastric region appears to be excessively rare. There is no mention of it in Konjetzny's article on hyperplastic tuberculosis of the stomach in Henke and Lubarsch's *Handbuch* (1928), nor can we trace another case of the kind in the literature.

The exact histological diagnosis in the present case has proved difficult. For the most part the lesion is non-specific, while presenting a widely different picture from that of ordinary peptic ulceration. There are, however, a number of follicular structures with Langhans-type giant cells, which we are prepared to accept as tubercle follicles even in the absence of demonstrable acid-fast bacilli. They are scattered throughout the more cellular part of the lesion, often in close proximity to small blood-vessels, though themselves avascular. This difficulty in firmly establishing the histological diagnosis of tuberculosis is well recognized in the hyperplastic lesion of the ileo-cæcum, where a number of blocks may have to be cut before a typical tuberculous field presents itself. Nor is it necessary that tubercle bacilli should be demonstrated before a reasonably probable diagnosis is ventured. By analogy with the intestinal disease, as well as with the published cases of hyperplastic tuberculosis of the pylorus, the lesion in this case is probably primary.

The possibility of the lesion being syphilitic was considered and rejected. There was no history of syphilis and none of the stigmata of that disease; the Wassermann reaction was negative, and the histological characters were not at all suggestive.

3. Not the least interesting feature of this case is the fact that the whole of the upper loculus, except a narrow, bright-red zone of surviving mucosa along the lesser curvature, is completely lined by squamous epithelium. Accompanying

histological changes are great hypertrophy of the muscularis mucosæ, which is everywhere present beneath the epithelium, and dense fibrous thickening of the submucous coat. The squamous differentiation stops short at the stage of stratum lucidum. Whether this is a genuine metaplasia due to the changed environment resulting from the mid-gastric obstruction, or whether it has arisen by squamous epithelial replacement extending down from the œsophagus, it is perhaps idle to speculate. The condition simulates closely the normal state of affairs in the stomach of rodents and some other animals, and might reasonably be designated 'rattisation' of the stomach.

Clinical.—The chief interest centres in the diagnosis and in the possibility of cure by surgical removal. It would seem that a clinical diagnosis of carcinoma can hardly be avoided in these cases. Even at operation it may well be impossible to go beyond this. As bearing on the possibility of complete eradication, it may be mentioned that a large proportion of reported cases of hyperplastic tuberculosis of the pylorus has been regarded as primary. Of twenty-four cases of tumour-like tubercle of the stomach collected from the literature by Leriche and Mouriquand (1909), two only presented evidence of a clinically demonstrable lesion in some other organ. This is in accordance with the known facts of hyperplastic tuberculosis of the ileo-cæcal region.

In the present case, complete eradication of the disease by the operation of total gastrectomy would seem to have been effected. Both the immediate result and the after-history (during nine months) have been wholly satisfactory.

We are indebted to Dr. H. B. Scargill for the X-ray photographs.

BIBLIOGRAPHY

- COLLINSON, H., and STEWART, M. J., *Brit. Jour. Surg.*, 1927-28, xv, 626.
 KONJETZNY, G. E., in Henke and Lubarsch's *Handbuch der speziellen pathologischen Anatomie und Histologie*, 1928, vol. iv/2, 1048. Berlin.
 LERICHE, R., and MOURIQUAND, E., *Rev. de Chir.*, 1909, xxxix, 520.

HALLUX VALGUS: A COMPARISON OF THE RESULTS OF TWO OPERATIONS

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THE respective merits of the two principal operations for hallux valgus have been argued for many years. Surgeons are still divided and are inclined to put forward their own personal impressions unendorsed by the patient.

In order to get new light on the question I determined some years ago to perform the two operations upon examples of bilateral hallux valgus, and these have now been under observation long enough to justify some conclusions. It has throughout been my intention that the final pronouncement should be made by the patient and not by the surgeon, since the patient is a much better judge of his pain (and its relief) than the most sympathetic observer. It ought, moreover, to be especially easy to get an opinion on the relative comfort of two operated toes in the same patient.

An experiment such as this would be unjustified at the hands of anyone who believed in the sole efficacy of one operation. Past experience had shown that excision of the metatarsal head gave excellent results in the majority of patients and after a sufficient time. This opinion was supported by Perkin's analysis of the results of the operation on more than 50 patients.¹ Nevertheless, I was assured by surgeons whose opinion I respected that excision of the base of the proximal phalanx was a much better operation, and from a casual perusal of recent literature it seemed that this view was gaining ground,² though evidence to support it was entirely lacking.

OPERATIVE PROCEDURES

The following procedure was adopted. Examples of hallux valgus needing a bilateral operation were dealt with differently on the two sides by standardized operations.

1. Excision of Head of First Metatarsal (Fig. 239).—A tourniquet was applied and a curved incision with its base downwards was made over the inner aspect of the first metatarso-phalangeal joint. A circular area of bursa and capsule the size of a threepenny bit was then removed and the joint exposed and cleared above and below. The head and neck of the metatarsal were removed by an osteotome, and the shaft was trimmed with bone forceps and smoothed with a file after removing all loose fragments of bone. The skin was closed with sutures and the toe held in a corrected position by a zinc-gelatine ('viscopaste') bandage.

2. Excision of Base of First Phalanx and Removal of Metatarsal Exostosis (Figs. 240, 241).—A tourniquet was applied and a dorsal incision made over the metatarso-phalangeal joint, which was then exposed on its dorsal, inner, and outer aspects. The base of the first phalanx, together with a small part of its

shaft, was then removed with an osteotome or bone forceps. The exostosis on the inner aspect of the first metatarsal was next removed with an osteotome, and the roughened parts of both bones smoothed with a file after extracting all loose fragments of bone. The articular and weight-bearing surfaces of the metatarsal were not damaged except in so far as removal of the exostosis encroached upon them. The skin was closed and the toe held in a corrected position by a zinc-gelatine ('viscopaste') bandage.

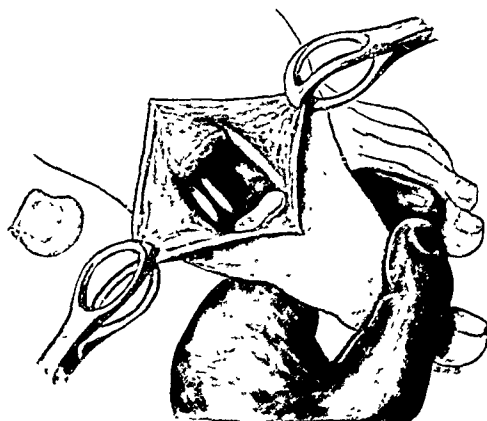


FIG. 239.—Left hallux valgus. Operation for excision of head of first metatarsal. The head of the bone has been removed, and is shown separately. The extensor hallucis longus tendon and the sesamoid bones are seen.

After-treatment was the same for both toes. The bandage was cut off after ten days and the stitches removed. A fresh 'viscopaste' bandage was applied for another week or ten days, and the patient encouraged to begin walking in a loose shoe or felt slipper. In most instances the shoes were wedged one quarter of an inch on the *outer* border in order to encourage weight-bearing on the affected part of the foot and to diminish any tendency to secondary metatarsalgia.³

Although many operations have been advocated for hallux valgus these are the two in common use. The description given is merely that of the ordinary procedure with no special additions except the use of a 'viscopaste' bandage. The sesamoid bones are not removed in either operation and are in fact left severely alone.

COMPARISON OF RESULTS

At the close of the period of observation, which has varied between one and three years, the patient was asked for a report. It was made clear that inquiry was solely directed to comfort, and each patient was asked to indicate whether the toes were equally comfortable or to state which one was the better. The results are set out in *Table I*, and furnish, as far as I am aware, the first attempt to compare the result of two different operations on the same patient. It may be again stated that the verdict is the patient's, and refers solely to the relief of pain one to three years after the operations.



FIG. 240—Right hallux valgus. Excision of base of first phalanx and removal of metatarsal exostosis (Stage 1). The base of the phalanx has been removed, and is shown separately. The exostosis on the inner surface of the metatarsal has not yet been removed.

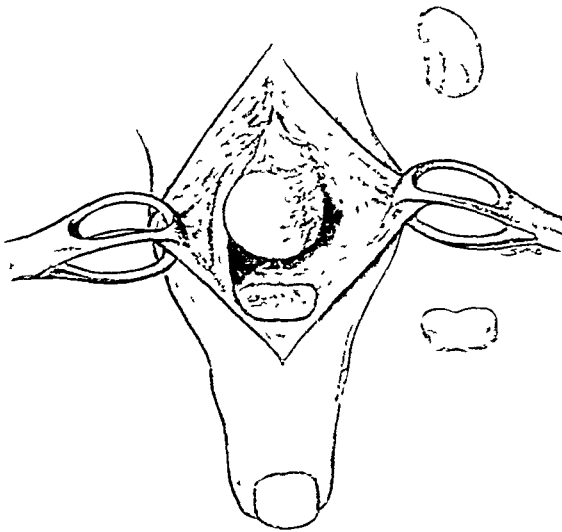


FIG. 241—Right hallux valgus. Excision of base of first phalanx and removal of metatarsal exostosis (Stage 2). The exostosis on the inner surface of the metatarsal has now been removed, and is shown separately in the upper part of the figure.

Table I.—COMPARISON OF OPERATIVE METHODS IN TWENTY CASES OF BILATERAL HALLUX VALGUS ON BASIS OF PATIENTS' FINDINGS

CASE No.	INITIALS	SEX	AGE	PERIOD OF OBSERVATION SINCE OPERATION	PREFERS SIDE ON WHICH METATARSAL HEAD EXCISED	PREFERS SIDE ON WHICH BASE OF PHALANX EXCISED	FINDS NO DIFFERENCE BETWEEN TWO SIDES
1	G. H.	F.	20	2½ yr.			×
2	A. D.	F.	46	2¾ yr.	×		
3	B. R.	F.	34	2½ yr.			×
4	D. G.	F.	45	2½ yr.			×
5	A. E.	M.	59	3 yr.			×
6	R. D.	F.	18	3 yr.			×
7	P. S.	M.	19	Nearly 2½ yr.			×
8	F. C.	F.	42	3 yr.			×
9	N. R.	F.	21	2½ yr.	×		
10	P. W.	F.	24	2½ yr.		×	
11	A. C.	F.	67	2 yr.			×
12	C. S.	F.	44	2 yr.		×	
13	R. W.	F.	24	2 yr.			×
14	D. S.	F.	25	1¾ yr.			×
15	R. M.	F.	32	1¾ yr.		×	
16	M. M.	F.	49	1½ yr.		×	
17	L. G.	F.	28	1½ yr.			×
18	L. P.	F.	52	1½ yr.		×	
19	M. D.	F.	34	1 yr.	×		
20	V. M.	F.	25	1 yr.	×		
				Total ..	4 (20%)	5 (25%)	11 (55%)

DISCUSSION

The series, though small, represents the verdict of twenty patients on forty operations, and gains some value from its uniformity of technique.

If one toe was more painful than the other it was not necessarily treated by the same operation. The metatarsal head was excised eight times on the right side and twelve times on the left. Metatarsalgia sometimes exists before operation, and Perkins¹ has rightly insisted that it is not relieved by operating on a coexisting hallux valgus. I would go further and add "even when the hallux valgus was the original cause of the metatarsalgia". It is equally true that some patients induce

metatarsalgia after operation by persistent refusal to bear weight on the inner border of the foot. They may thus spoil the functional result of a successful operation and even select the wrong toe as being the more comfortable by erroneously attributing the metatarsalgia to the operation. I believe that this may have occurred in my series, but it is impossible at this stage to make allowance for it. It probably has not affected the majority, since it is noteworthy that more than half the patients report both toes equally comfortable from one to three years after operation.

SUMMARY

Twenty patients suffering from bilateral hallux valgus were subjected to different operations on the two sides. On one side the metatarsal head was excised, and on the other the base of the phalanx and the metatarsal exostosis.

From one to three years elapsed after the operations, and the patients were then asked to choose between the two toes purely from the point of comfort. The results are set out in a table, and seem to indicate that there is nothing to choose between the two operations as a means of relieving the pain of hallux valgus.

REFERENCES

- ¹ PERKINS, G., 'Removal of the Metatarsal Head for Hallux Valgus and Hallux Rigidus', *Lancet*, 1927, 1, 540.
- ² LAKE, C., *The Foot*, 1935, 188. London: Baillière, Tindall & Cox.
- ³ BANKART, A. S. B., *Manipulative Surgery*, 1932, 56, 57. London: Constable & Co.

PSEUDOTUBERCULOMA SILICOTICUM

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THE reaction of the body tissues to the implantation of foreign bodies has been extensively investigated. Unabsorbable ligatures have been shown to produce endothelial and giant-cell reaction with fibrosis. The effect on the lungs of inspired irritating particles such as coal, asbestos, and silica dusts is of great importance in industrial diseases. It is seldom, however, that the implantation of undetected foreign particles forms a tumour which presents itself as a clinical and pathological problem. S. G. Shattock (1916-17)¹ published such a case, and gave the name of pseudotuberculoma silicoticum to the condition in view of the close resemblance of the tuberculous granuloma to the tissue reaction to buried particles of silica in a wound. J. S. Faulds (1935)² published two further examples of the condition. In all three cases the foreign particles were demonstrated in sections by means of the polarizer.

The following additional case came under our observation in the Casualty Department of University College Hospital.

A girl of 22 years complained of a tender nodule underlying an old scar on the left forearm. The nodule had developed slowly during the previous three months. The scar was the result of a fall sixteen years previously while walking along a flinty road. The district has much sandstone in it, and after a rainstorm a deposit of sand can be seen on the road. She complained that the nodule was painful on resting her arm on the elbow or on knocking it against anything.

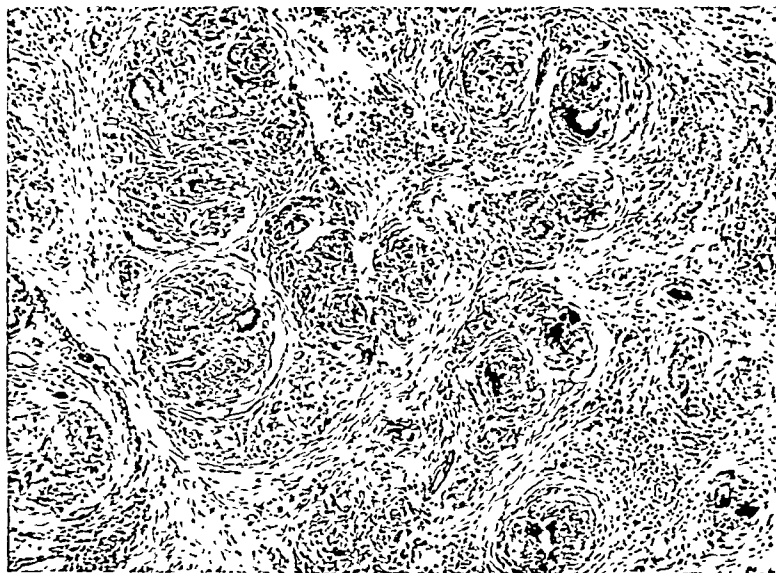


FIG. 242.—General low-power view ($\frac{1}{2}$ in.) of a piece of a section showing multiple giant-cell systems and a tear in the section. ($\times 78$.)

Examination revealed a scar $\frac{3}{4}$ in. long and $\frac{3}{8}$ in. wide, lying transversely across the back of the forearm 2 in. distal to the tip of the olecranon. Underneath the scar was a tender nodule the size of a hazel-nut, firm and slightly fluctuant, and attached to the scar and the deep fascia. The diagnosis of implantation dermoid was made, and it was proposed to excise the scar together with the cyst and to leave a longitudinal linear scar.

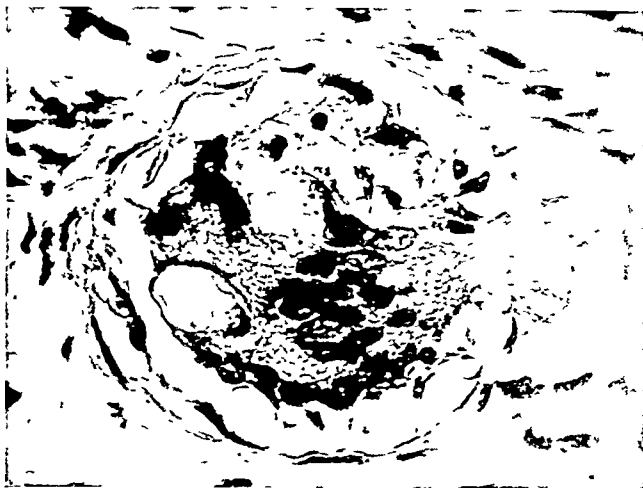


FIG. 243.—High-power view ($\frac{1}{2}$ in, oil immersion) of a giant cell with embedded silica particle (500)

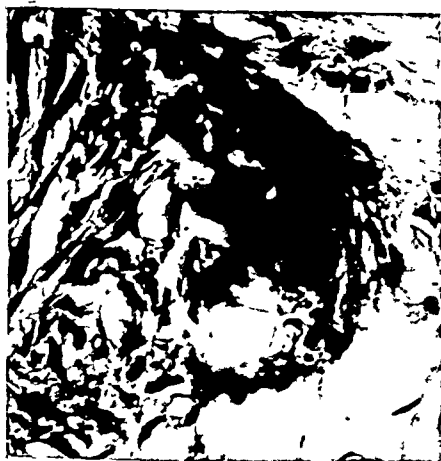


FIG. 244 —High-power view ($\frac{1}{2}$ in, oil immersion) of a giant cell with embedded silica particle. ($\times 360$.)



FIG. 245 —The same giant cell as in Fig. 244, showing the crystal under the polarizer ($\times 360$)

At operation a small bursa was found superficial to the nodule, which was fibrous and densely adherent to the capsule of the elbow-joint, from which it had to be dissected.

Serial sections at 50- μ intervals were cut through the nodule, which proved to be extremely hard and gritty and took several chips out of the edge of the razor. The sections were torn and ragged, but no foreign particles were observed with the naked eye. The

microscopic appearance was almost exactly that of a tuberculoma. Clumps of large irregular giant cells, many with peripheral rings of nuclei, were scattered throughout the field, surrounded by epithelioid cells and lymphocytes and fibrous trabeculae (*Fig. 242*). There was no sign of caseation in any of the sections. Ziehl-Neelsen staining revealed no tubercle bacilli. On carefully searching through the sections with the high-power lens, several crystalline particles were observed, some embedded in the fibrous tissue, others actually within the giant cells (*Figs. 243, 244*). The sections were therefore examined with polarized light and the particles were mostly found to be doubly refractile (*Fig. 245*). Unfortunately there was insufficient material left for chemical analysis. The wound healed by primary intention, and three weeks later there was no evidence of recurrence and the scar was not tender. A radiogram of the forearm at this time showed no foreign bodies.

COMMENTARY

In the four cases which have been reported, three have given a definite history of trauma, and in the fourth a particle of mineral, later identified as Welsh slate, was found in the centre of the tumour, conclusive evidence of some previous accident. Ours was, however, the only one in which a scar was obvious clinically. The time of onset of symptoms is interesting in view of the long period of apparent quiescence between the infliction of the injury and the appearance of the lump. In Shattock's case the interval was eleven years, in one of Faulds' it was ten years, and in ours sixteen years. Faulds attempted to explain this in his case by a second trivial accident three months before the onset of symptoms, which he suggested may have driven some of the particles through their fibrous capsules and so reactivated the cellular proliferation. In two of the cases the nodule was situated on the forearm, and in two on the face, the parts normally uncovered and exposed to injury. In the three cases in which an accident was remembered, it occurred on the road, an obvious source of silica.

The clinical appearance of a nodule underlying the skin of the exposed parts and associated with previous trauma simulates that of an implantation dermoid. In two of the cases this diagnosis was actually made. The nodules, however, appeared solid in all except our case, in which the fluctuation obtained was shown to be produced by an adventitious bursa. In addition, it was definitely tender in two cases, an uncommon finding in implantation dermoids. These points taken in conjunction with the history of a road accident many years previously are useful in the differential diagnosis, and make it advisable to bear the condition in mind and to take a radiogram of the part. Further proof of the condition is afforded by the detection of foreign particles with the naked eye, and in the tearing of the sections and the chipping of the razor during cutting, which was present in one of Faulds' cases as well as in ours.

The microscopic appearance closely simulates that of a tuberculoma. If tubercle bacilli can be demonstrated by Ziehl-Neelsen staining, it is of course valuable evidence against the diagnosis of pseudotuberculoma, but a negative finding proves nothing. In our case many of the giant cells had ringed nuclei, and it was impossible to exclude tubercle by the ordinary histological methods. The complete absence of caseation, however, was significant, and this was also seconded in the previous cases. In Shattock's case and in ours suspicious-looking particles were observed embedded in the giant cells and fibrous tissue, and on careful focusing it was possible to make out their crystalline nature. It is probable that the multiple groups of giant-cell systems correspond to individual particles of embedded silica.

The final evidence is afforded by means of polarization, and the particles were readily picked out in all the cases. Faulds in his article pointed out numerous sources of error in this method, and showed how collagen fibres, keratinized epithelium, striate muscle, and erythrocytes were all capable under certain conditions of refracting polarized light. In one of Faulds' cases a sufficiently large particle was obtained for chemical analysis, and the evidence of the presence of silica was therefore quite conclusive in this case.

It is evident that, although clinically this condition may simulate an implantation dermoid and pathologically a tuberculoma, when the evidence is taken as a whole the clinical history and pathological findings are typical and conclusive.

SUMMARY

1. Reference is made to three previously recorded cases of pseudotuberculoma silicoticum, and a fourth is described.

2. The clinical features of the condition are the history of a fall on a road and the development of a solid tender nodule at the site of injury many years later. The differential diagnosis from implantation dermoid is made.

3. *Pathologically the condition is characterized by a fibrous gritty tumour, closely resembling a tuberculoma microscopically, but without caseation.* Crystals of silica can be picked out with the high-power lens and polarized light, and silica may be found on chemical analysis.

We are indebted to Dr. G. R. Cameron for his advice and co-operation.

REFERENCES

- ¹ SHATTOCK, S. G., *Proc. Roy. Soc. Med. (Pathol. Sect.)*, 1916-17, x, 6.
² FAULDS, J. S., *Jour. Pathol. and Bacteriol*, 1935, xli, 129.

EMBOLECTOMY ON THE VESSELS OF THE EXTREMITIES*

BY EINAR KEY, STOCKHOLM

ONE of the most satisfactory operations that can be performed is the removal of an embolus by means of arteriotomy (embolectomy) in suitable cases. This has been made possible through the development of the technique of operations on the vessels of the human body.

HISTORICAL REVIEW

The first to try to remove an embolus by arteriotomy was Ssabanejew (1895). Because of threatened gangrene in one leg as a result of embolism he tried to remove the embolus from the femoral artery, but was not successful. During the first decade of this century, several surgeons tried in vain to remove an embolus or an arterial thrombus by arteriotomy in order to restore the circulation and prevent development of gangrene. Among the first to attempt this, the names of Moynihan, Francis Stewart, Sampson Handley, and Gordon-Watson must be mentioned. Also I wish to call to mind the interesting case operated upon by Murphy in 1908.

Trendelenburg hit upon the daring and ingenious idea of operating upon the pulmonary artery to remove large emboli producing fatal symptoms. In experiments on calves he produced artificial pulmonary emboli by introducing long pieces of organic tissue into the jugular vein and then succeeded in removing these by pulmonary arteriotomy. In 1907 Trendelenburg first tried to remove a pulmonary embolus from a patient. The case was that of a woman, 70 years of age, with sudden collapse due to a pulmonary embolus. He was successful in removing the embolus, but the patient died of hæmorrhage. Several surgeons then attempted to do Trendelenburg's operation for threatening pulmonary embolism, but it was a long time before a good result was attained, and doubt began to be cast upon the practical significance of the operation. Justifiable attention was therefore awakened when at the German Surgical Congress in 1924 Kirschner reported a case he had been able to save by means of Trendelenburg's operation. This fortunate experience led to renewed interest in the operation, and successfully operated cases have since been reported by Meyer, Giertz, Crafoord, and Nyström. However, it would seem to be only exceptional cases where this operation can save patients with severe symptoms of pulmonary embolism.

Embolectomy is of greater importance for the removal of emboli producing dangerous circulatory disturbances in the extremities. Even here, however, it was a long time before good results were attained. In 1911, at a meeting of the Société de Chirurgie de Paris, Lejars stated that there existed little hope that the removal of an embolus would actually do away with the obstruction it had produced and

* A lecture recently delivered at the London Post-graduate School of Medicine.

that a new thrombus would not develop. Shortly after, in the same year, Labey performed the first successful operation for embolus. It was situated in the common femoral artery and there were symptoms of impending gangrene. The operation was done six hours after the embolism developed. The second such case with a successful outcome was operated upon by me shortly after. This was also an instance of embolism of the femoral artery operated upon six hours after the onset of symptoms.

More and more cases have come to operation. At the Maria Hospital in Stockholm we have devoted the greatest interest to this operation. From 1912 to 1934, 382 embolectomies have been performed in Sweden on cases where the emboli have given rise to grave circulatory disturbances in the extremities. At the Maria Hospital, and in my private practice to date, 48 embolectomies have been performed on 43 patients (there was an embolus in both legs in 5 cases). I myself have done 32 embolectomies on 30 cases.

ETIOLOGY

A thrombus in the heart is the commonest source of an embolus giving rise to dangerous circulatory disturbances in the extremities. More rarely the origin is from a thrombus in the wall of the aorta. The cause of thrombus formation in the heart with subsequent embolism in the above-mentioned sites is usually a decompensated mitral valvular lesion, and occasionally myocarditis. Sometimes these emboli develop following an infectious disease, while others appear shortly after an operation on a patient who has never had any symptoms of cardiac failure. In these instances also the source of the embolus is probably a cardiac thrombus. In rare cases paradoxical embolus may occur.

Women seem to have emboli in the extremities oftener than men.

In the age group 31-40 there is a great increase in the frequency, and most of the cases are between 31 and 70 years of age.

LOCALIZATION

An embolus will lodge most readily where a vessel divides. In the greater number of cases that have come for operation the embolus has been located at the division of certain large vessels, such as the aortic bifurcation, parts of the iliac vessels, the femoral artery, the popliteal artery, and the axillary where the scapular artery branches off.

The localization of the embolus in the 382 Swedish cases (collected by Nyström) can be seen in *Table I*.

Thus, most of the emboli operated upon were situated in the lower extremities, and then most often in the common femoral,* with the second greatest frequency in the common iliac. In some cases the embolus which was the cause of the operation had been preceded by other emboli in a site which did not demand surgical interference. After the operation, fresh emboli have sometimes appeared. *It is important to realize that not infrequently two or more emboli requiring operation*

* Throughout this paper the term common femoral artery is used to refer to the portion of the femoral artery between the external iliac and the origin of the deep femoral; from here to the popliteal artery it is regarded as the superficial femoral artery.

may appear at different sites. They may be situated either in the main artery of the same extremity or in another.

Table I.—LOCALIZATION OF EMBOLUS IN 382 OPERATED SWEDISH CASES

ARTERY	NUMBER	FREQUENCY
		per cent
Axillary }	45	11·8
Brachial }		
Ulnar	1	0·3
Bifurcation of aorta ..	17	4·5
Iliac	66	17·3
Femoral	208	54·5
Popliteal	43	11·3
Tibial	2	0·5
Total ..	382	100

SECONDARY THROMBOSIS AND GANGRENE

An embolus not removed generally in time goes through a stage of secondary thrombus formation. As a result, an embolus which at first did not fully block the lumen may become an obstruction. The embolus increases in size most easily and for the most part towards the periphery, but it may also extend in a centripetal direction. The secondary thrombosis impedes the collateral circulation, and in this way the danger of gangrene is increased. The danger of gangrene is therefore greater with embolism than in the case of ligature of the vessel at the corresponding site. The following table provides some figures on the percentage frequency of gangrene following ligature of certain vessels.

Table II.—FREQUENCY OF GANGRENE AFTER ARTERIAL LIGATURE

SITE	WOLFF, 1908	HEIDRICH, 1921	MAKINS, 1922
	per cent	per cent	per cent
Upper extremities ..	7·8	5·9	—
Lower extremities ..	20·4	20	—
Subclavian artery ..	4·8	9·7	—
Axillary artery ..	15	9·8	—
Brachial artery ..	4	3·13	—
Aorta	—	100	—
Common iliac artery ..	50	100	—
External iliac artery ..	11·2	13·4	16·6
Common femoral artery ..	25	21·8	25·9
Superficial femoral artery	12·7	10·4	14·1
Popliteal artery ..	14·9	37·2	32·5

This table shows that there is more danger of gangrene in ligature of the great vessels of the leg than of the arm. Experience teaches that when an embolus is located at the branching of the above-mentioned arteries there is more or less pronounced danger of gangrene in the extremities. Experience has also shown that

with an embolus at the aortic bifurcation, in the common iliac artery, the common femoral, and the popliteal, there is more danger of gangrene than with an embolus in the axillary artery and in the upper portion of the brachial.

The time of the appearance and spread of secondary thrombosis varies considerably. The danger generally increases with the time elapsing after the embolus appears, yet the increase of the risk is not in direct proportion to the length of time. I have seen secondary thrombosis beginning within two hours after the appearance of an embolus. Sometimes, however, in a case of late operation there has been no thrombosis. In a case operated upon by Ipsen there was no secondary thrombosis after eleven days. However, in one of Sundberg's cases a thrombus no less than 86 cm. in length had formed within twelve hours. A secondary thrombus renders the operation more difficult and the prognosis more unfavourable.

In cases which come early for treatment, it is generally easy to distinguish at operation between the original embolus and the secondary thrombus formation; in late cases this may be difficult or impossible. In some cases it may also be difficult or impossible to determine where the primary embolus was located.

SYMPTOMS

The symptoms may set in either suddenly, which is most usual, or slowly. In the former case, the embolus is so large that it causes serious disturbance of the circulation. In the latter case it at first obstructs only a part of the lumen. Later, by secondary thrombus formation, or by the appearance of a new embolus, the lumen may become blocked, and the symptoms consequently increase. The degree of circulatory disturbance caused by the presence of an embolus depends upon several factors: (1) whether it fully obstructs, (2) the development of the collateral circulation, (3) the secondary thrombus formation, (4) the condition of the walls of the vessels, and (5) the action of the heart. Consequently the intensity and extent of the circulatory disturbance vary between quite mild cases without any danger of gangrene and threatening gangrene of varying extent.

The symptoms characterizing an embolus are partly subjective and partly objective. *The subjective symptoms* are pain, a sensation of cold, and disturbances of sensibility. *The objective symptoms* are change in colour of the skin, a lowering of skin temperature, disturbed motility, absence of skin- and tendon-reflexes, and absence of pulsation.

The pain most often sets in suddenly, and this is highly significant. At the same time, the patient experiences a sensation of cold and numbness in the extremity affected. The sensory disturbances vary from partial to complete suspension of sensory function. The boundary of complete suspension is not often sharply defined, but the transition to partial suspension may be clearly observed.

The suspension of circulation results in marked anæmia of the affected extremities, the temperature falls, and the skin becomes deadly pale or possibly somewhat cyanotic. Discoloration may be seen in livid to dark blue patches. As one would expect, the anæmia and low temperature are most pronounced towards the periphery. A prick from a needle causes no bleeding. The motility is suspended or restricted in proportion to the degree of circulatory disturbance.

The sensory disturbances, the change in the colour of the skin, and the cool part of the extremity do not always fall quite within the same boundary, neither

do they always quite correspond to those areas where the motility has been affected. Apparently motility at least may remain for some time in a part of the extremity which is cold and in which sensory functions are suspended or impaired.

There is usually no pulse below an embolus. One can sometimes palpate an embolus like a string in the painful area. The possibility of palpating an embolus depends upon its situation and on the corpulence of the patient.

As the collaterals take over a part of the circulation, the limits of the region threatened with gangrene will be situated at a longer or shorter distance peripheral to the embolus, and they are not at first very sharply defined. The extent of the region threatened with gangrene varies in different cases, as has been said, according to the development of the collaterals, the size of the embolus, the heart action, the condition of the vessel walls, and also the stage reached by the secondary thrombosis. Likewise, the symptoms of circulatory disturbance may diminish or disappear should an embolus in a suitable position become again detached and lodge more peripherally. If, from being not quite obstructive, an embolus becomes so through secondary thrombus formation or through blocking the collaterals, the symptoms naturally increase.

Some cases have precursory symptoms probably depending upon a small embolus. In other cases, pronounced symptoms in another area have preceded an embolus. It would take too long to go into the symptomatology of emboli in various locations.

DIAGNOSIS

The symptoms of an obstructing embolus that appears suddenly in a large vessel of the extremities are so marked (sudden severe pain in the extremity, a feeling of cold and numbness, sensory and motor disturbance, and disturbance of the circulation) that the diagnosis is seldom difficult. When the embolus is not entirely obstructive to begin with, but becomes so gradually by augmentation, the symptoms are often not so pronounced at first, and the diagnosis may be difficult.

Differential Diagnosis.—

Thrombus due to Arteritis.—It is of the utmost importance to distinguish between an embolus and an arterial thrombus due to arteritis. Indicative of embolism are sudden onset of the symptoms in a patient with organic disease of the heart or with an active predisposition to thrombus formation as a result of an infectious illness or an operation. The origin of an embolus, however, cannot always be pointed out. Often these patients have had, or have, circulatory decompensation. When the symptoms do not set in so quickly the differential diagnosis is more difficult. In the case of an arterial thrombus the patient has often had premonitory symptoms for a long time, even for years—sensations of cold and numbness, neuralgia or rheumatic pains in the extremity, and circulatory disturbance in the peripheral parts of the limb. Furthermore, one must of course take into consideration the presence of arteriosclerosis. It is true that the evidence of arteriosclerosis is not decisive and does not preclude embolism.

Developing Thrombus.—Mistakes have been made between a developing thrombus and an embolus. With a thrombus in the vein, the extremity is warm, cyanotic, and swollen. As a rule the pulse is perceptible and the sensibility unchanged, and usually the thrombosing vein feels distressingly tender. With an embolus,

the extremity is cold, generally pale, sensibility is partially or completely suspended, and the pulse is imperceptible or very weak. The thrombosing vessel can sometimes be palpated. When a thrombus precedes an embolus, the latter may be easily overlooked.

Local Traumatic Arterial Thrombosis.—After trauma with a slight internal injury, without the wall of the vessel being ruptured, an arterial thrombus may appear in the injured place. As a rule, the differential diagnosis in such a case can be made from the history.

To Localize an Embolus.—As already shown, owing to the collaterals, the circulatory disturbance will appear peripherally and only at a greater or less distance from the embolus. In many cases, therefore, the conclusion has been reached that the embolus was seated still more peripherally. By taking into consideration the fact that an embolus generally lodges at the division of an artery, and that it is seated more or less central to the boundary of the circulatory disturbance, and also by carefully observing where the pulse ceases in the artery in question, one can generally succeed in localizing it. When taking the pulse, one must take into consideration that a difficulty in feeling it may be owing to the patient's weak heart action.

SOME POINTS AFFECTING THE OUTLOOK

Injury to the Intima with Consequent Thrombus Formation.—In the area where an embolus is situated, a lesion of the intima is likely to develop sooner or later, and, after the embolus has been removed, may cause thrombus formation. The vessel may become obstructed, and the result of removing the embolus come to nothing. There is no doubt that it is difficult, if not impossible, to decide in a given case whether a thrombus which has arisen after the removal of an embolus was caused by a lesion of the intima or by some mistake in technique. Even though the risk of the lesion with its consequent tendency to thrombus formation increases with the length of time from the occurrence of the embolism, yet the increase of the risk is not in direct proportion to the length of time; wide variations and strange exceptions are to be found just as in secondary thrombus formation.

Relation of Length of Time of Obstruction to Tissue Vitality.—Naturally of great interest and practical importance is the question *how long the blood-stream through the main arteries to an extremity can be suspended by an embolus without injury to the vitality of the tissue.* As yet it is not known with exactitude how long the blood-stream to an extremity can be obstructed without loss of vitality in the area supplied. It is general knowledge that, if one must bandage an extremity to stop hæmorrhage, for example with an Esmarch, one must not leave it in place too long, or ischæmic contraction or even gangrene will appear. It is stated that ischæmic contraction may arise when the bandaging has lasted from two to three hours. In the case of an embolus, the conditions are quite different. Here the arterial circulation is eventually suspended while the vein and lymph channels are not obstructed. Nor are the nerves exposed to pressure. Furthermore, the embolus may happen not to be fully obturating, until it becomes so by a secondary growth, so that the time when the symptoms set in, and the time of complete suspension of the flow of blood through the arteries, do not

coincide. In the case of an embolus, the collateral channels also play more or less of a part, and the heart action and condition of the vessel walls are of considerable importance. The patient's general condition and the vitality of the tissues likewise play their part.

After the removal of the chief embolus, small emboli that have previously lodged further out in the peripheral arteries may interfere with the circulation. Secondary thrombosis may obstruct important collateral channels. After the removal of the embolus, if the secondary thrombus has not also been completely taken away, more or less circulatory disturbance will be caused. There are thus many factors contributing to the result. No set time before gangrene occurs can be given; one must be prepared for great individual differences. Twenty-four hours was the longest time that has passed between the appearance of the symptoms and embolectomy, without ischæmic necrosis or gangrene setting in. Probably the circulation was not completely obstructed so long as that.

EMBOLECTOMY

Anæsthesia.—In all my cases I have used local anæsthesia with novocain and adrenaline, which gave a complete and satisfactory anæsthesia. As these operations have to do with individuals of low physical tone, and mostly with irregular heart action, it is most important that the operation be carried out as carefully as possible. The risk in operation is, no doubt, considerably less if it can be done with a local anæsthetic. When operating on the relatively easily accessible vessels, such as the lower part of the axillary, brachial, external iliac, femoral, and popliteal arteries, one generally ought to manage with local anæsthesia. In removing an embolus in the aorta, or in the common iliac artery, if one must do a laparotomy, it will as a rule be necessary to use a narcotic.

Technique of Operation.—When performing embolectomy, it is of highest importance that the right technique be used. The slightest mistake may cause thrombosis and thus the good result of the operation may be lost.

As the technique for the suturing of vessels is nowadays well known, I shall not give an account of it. I myself have largely used Carrel's technique, with very fine needles and very fine silk sterilized in vaseline. In my first cases, I covered the wound with vaseline compresses. In this procedure the rubber gloves used during the operation become very slippery, which makes the sewing with fine needles considerably more difficult. I have therefore made a slight change in the technique. As sodium citrate prevents the blood from clotting, I have replaced the vaseline compresses with compresses dipped in 2 per cent sodium citrate. The arteries have been surrounded with the same, and the surface of the wound covered with it. From the time when the vessel is opened until the completion of suturing, instruments and gloves are rinsed in 2 per cent sodium citrate solution. The curettes or probes that are used for removing the embolus should be smeared with vaseline.

When the artery is laid bare, the operator satisfies himself as to the position of the embolus and its extension proximally and peripherally. If, as is usually the case, the embolus has lodged at the division of an artery, he carefully attempts to isolate the vessel where it divides so that flexible artery clamps may be put upon these also. Should other branches of the artery lead from the area in which the

embolus is situated, the most prudent method is to isolate these vessels as well, in so far as vessel clamps or rubber tube can be used. When the artery has been surrounded with compresses dipped in sodium citrate solution, a weak vessel clamp is placed central to the embolus. Instead of pinching the artery by a soft vessel clamp I have produced a stoppage by making a kink in the vessel by pulling on a thin rubber tube which has been drawn around the vessel.

When the arteriotomy has taken place the embolus may happen to fall out of itself or by a slight pressure on the vessel. Generally it must be lifted out, and this should be done exceedingly cautiously, with a fine forceps or a small blunt curette, in order that the intima may not be injured. At times the embolus is so fragile that it goes to pieces when taken hold of and has to be removed in many parts. When the embolus is long, or a secondary thrombus is present, it may sometimes be difficult, or impossible, to clear entirely the vessel. It is of utmost importance that this be done as completely as possible. Should one not succeed in removing the secondary thrombus, and this is of such length that it cannot be extracted otherwise, one may use a blunt instrument through an arteriotomy opening below and attempt to loosen the embolus and let the flow of blood wash away the pieces of the thrombus. Of course all instruments intended to be used in the vessels must be carefully covered with vaseline.

When the localization of an obstructing embolus is unfavourable from the point of view of surgical access, as in the aorta, the upper part of the axillary artery, and common iliac artery, it has been tried to move the embolus to the nearest easily accessible place with the aid of blunt instruments introduced in the artery through an arteriotomy. In this case the incision in the artery is made below the embolus in the nearest convenient place. The embolus is loosened with a blunt instrument introduced through the arteriotomy, letting the flow of blood wash away the embolus to the place of the incision. When doing this the artery must be closed by a clamp or rubber tube just below the arteriotomy, so that pieces of the thrombus may not flush into the peripheral parts of the vessel. The pieces are squeezed out through the incision in the artery or are lifted out.

The distal part of the external iliac artery can easily be exposed extraperitoneally under a local anæsthetic. The proximal part will be more difficult, particularly with stout persons. The common iliac artery and the aortic bifurcation are exposed most easily by laparotomy. Bauer was the first to remove with success an embolus in the aortic bifurcation by this method. Few other such cases, where the embolus was removed successfully by laparotomy, have been reported. Usadel preferred to perform a direct embolectomy after having exposed the aorta and the common iliac artery retroperitoneally by a large incision. A direct embolectomy through a laparotomy or large retroperitoneal exposure, which must usually be performed with a general anæsthetic, is nevertheless a great strain on these patients, who are mostly of poor physical tone.

It is far less trying for them if one loosens the embolus with a blunt instrument introduced through an incision in the femoral artery, letting the flow of blood rinse it out. That an embolus in the aortic bifurcation can be advantageously removed in this way has been shown in one of my own cases. In an operation after this method upon an embolus situated where the aorta divides, the other side of the common femoral artery also ought to be exposed first and a vessel

clamp placed there, so that the pieces of the embolus which are loosened and possibly forced out to this side may be stopped by the vessel clamp and easily taken out afterwards. In order to attempt the removal of an embolus from the aortic bifurcation by means of retrograde probing, it would often be necessary to do this from both sides. Nyström has tried in a case of bilateral embolism of the iliac arteries and in cases of embolism of the aortic bifurcation to bring the emboli down to incisions in the femoral arteries beneath the inguinal ligament by a kind of milking applied directly to the artery. An incision was made along the inguinal ligament and the hand was introduced in the retroperitoneal space, making its way closely along the large vessels to the sacral promontory. By pressure with the fingers the embolus was felt to burst, and it was possible to dislodge it down the vessels and by an incision in the femoral artery to remove the masses.

After the embolus and possibly secondary peripheral thrombi are removed, vessel clamps also should be placed so that the bleeding of the collaterals may not impede the sewing-up of the wound in the vessel wall. These clamps should be placed peripherally to the arteriotomy opening, and on the vessels that lead from the opening; that is to say, between the part of the main artery where the central and the peripheral vessel clamps have already been placed. As a secondary thrombosis may rise proximally to an embolus, one should take off the proximal clamps for a moment or two before sewing the vessel wall together. Thus any centrally placed thrombus, or remains of the embolus, can be flushed out by the stream of blood. During this flushing-out process, the peripheral vessel-clamps must be in place, so that any possible parts of the embolus or the central thrombus may not flush into the peripheral parts of the vessel.

When the embolus has been removed, the blood should be allowed to spurt out through the arteriotomy opening in a vigorous stream with intermittent jerks synchronizing with the pulse. Should the blood flow without pulsation, we have an indication that the passage is not quite clear, and that there is some obstacle above. This obstacle must then be cleared away, either by retrograde probing, or by laying the vessel bare higher up and making a fresh arteriotomy. Thrombi left in the peripheral arteries will cause more or less severe circulatory disturbance according to their extent.

As already stated, embolism is often recurrent, and two or more emboli frequently appear in the main artery in the same extremity. During the operation it is therefore most important to direct special attention to the possibility of manifold emboli within the chief artery. If such be overlooked, the circulation will not be fully restored, and gangrene may set in. After the embolus and the contingent secondary thrombus are removed and the vessel has been sewn together, investigation must be made as to whether the circulation has been completely restored in the extremity. Should this not be the case, an examination must be made to discover whether more emboli are present, and if so these must be removed if possible.

Arteriosclerotic changes of the wall highly endanger the result of embolectomy. The intima, which is altered by the arteriosclerotic changes, is further hurt by arteriotomy, and new thrombi can be formed which soon stop the passage. Arteriosclerosis does not exclude a good result of embolectomy. In an 82-years-old woman Torell has done embolectomy on the femoral artery with good result.

RESULTS

Up to now we have at the Maria Hospital and in my private practice operated 48 times on 43 patients (in some cases several embolisms occurred). Naturally I consider the result fully satisfactory only when the patient remains alive and the circulation is completely restored. Several of the patients, however, succumbed to their serious illnesses, though in many of the cases we succeeded in restoring the circulation. The results are tabulated in *Table III*.

Table III.—RESULTS IN 48 EMBOLECTOMIES

SITE	NUMBER OF OPERATIONS	RESULT OF OPERATION					
		Good	Death shortly after Operation		Death within a Month		Gangrene
			Good Circulation	Circulation not Improved	Good Circulation	Gangrene	
Axillary and brachial arteries	4	4	—	—	—	—	—
Bifurcation of the aorta ..	2	1	1	—	—	—	—
Common iliac and external iliac arteries ..	10	1	1	—	1	1	3
Femoral artery	24	9	1	2	3	5	1
Popliteal artery	7	3	—	—	—	1	3
Posterior tibial artery ..	1	1	—	—	—	—	—
Totals	48	19	3	2	4	10	10
Percentage	100	39.5	6.2	4.2	8.3	20.8	20.8

In the column of *Table III* labelled 'good' are inserted those cases in which the circulation is restored, where the extremity is saved from gangrene, and where the patient is alive. Those patients operated upon for embolus often succumb to uncompensated heart disease though the circulation in the extremity in question is restored by the embolectomy. In deciding the value and estimating the results of embolectomy these cases must be taken into account. If the patient dies shortly after the operation the judgement of the result naturally will be difficult or impossible. The circulation may be restored, but if the patient had been alive longer, perhaps a secondary thrombus would have arisen at the site of the operation, which would have again stopped the artery. If the patient is alive longer and the circulation in the operated extremity is restored and functions well, then the embolectomy so far has succeeded, even if the patient succumbs to his serious malady. I have therefore collected in different columns of the table the cases which have died shortly after the operation (within twelve hours) and those which have died between twelve hours and one month after. In the first of these columns I have separated those cases where the circulation was improved from those where the circulation was not improved after the operation. In the other group (those who died between twelve hours and one month after operation) the cases have been divided with respect to (1) those in which the circulation was permanently restored by the operation, and (2) those in which disturbed circulation and gangrene occurred after the operation. In the last column those cases are gathered which

were alive more than one month after the operation and in which gangrene had occurred. In the greater number of these cases an amputation was done.

The results in the 382 Swedish cases collected by Nyström are shown in Table IV.

Table IV.—RESULTS OF EMBOLECTOMY IN 382 SWEDISH CASES

SITE	DISCHARGED SURVIVING		DIED IN HOSPITAL	TOTAL
	With Restored Circulation	After Amputation		
Axillary artery } ..	20 (=44·4%)	9 (=20%)	16 (=35·5%)	45
Brachial artery }	—	—	1	1
Ulnar artery ..	—	—	14 (=82·3%)	17
Aortic bifurcation ..	3 (=17·7%)	—	43 (=65·2%)	66
Iliac artery ..	10 (=15·1%)	13 (=19·7%)	133 (=63·9%)	208
Femoral artery ..	43 (=20·7%)	32 (=15·4%)	20 (=46·5%)	43
Popliteal artery ..	9 (=20·9%)	14 (=32·6%)	—	2
Tibial artery ..	1	1	—	2
Total ..	86 (=22·5%)	69 (=18·1%)	227 (=59·4%)	382

The result of embolectomy on the axillary and brachial arteries is better than on the great vessels of the lower extremities. It seems to me that this can be explained on the basis of better collateral paths in the arm. The danger in ligation of the axillary and brachial arteries is considerably less than after ligation of the femoral or popliteal artery. In obstruction to the circulation due to thrombosis after embolectomy it is therefore probable that it is the collateral circulation which saves the upper extremity oftener than the lower. In cases of circulatory disturbance threatening the condition of the lower extremities, the best results have been obtained in the embolectomies on the femoral artery, the next best on the popliteal, slightly less good on the iliac, and worst on the aortic bifurcation.

The prognosis as to the possibility of preventing by embolectomy the development of gangrene in connection with an embolus depends very largely upon how soon the operation takes place after the appearance of the embolus. The importance of early operation appears in my material: 34 operations have been performed within ten hours after the onset of the symptoms; out of these, 19 (55·8 per cent) have regained normal circulation. Out of the 32 cases which were operated upon at the clinic in Upsala, 50 per cent were sent home with restored circulation.

AFTER-RESULTS

In most cases, embolectomy has been performed on patients with some degree of organic heart disease. These patients may go through the operation, yet the cause of the trouble still remains, and they generally succumb to it sooner or later. Nevertheless, many have had relatively good health for a number of years, and could do more or less work. Naturally the prospect of a lasting benefit is better in the cases where there has been no previous organic heart disease but the embolus is due to some other cause, such as infection.

Strömbeck has followed up 61 cases which up to 1933 were reported from Swedish hospitals surviving after embolectomy and with restored circulation. He

found that out of these about three-quarters still are alive 1 year after the operation, half of them 3 years, a third 5 years, and an eighth 10 years. Hindmarsh and Sandberg have followed up the patients from the Maria Hospital who were sent home with restored circulation. Though after two to three years most of the patients are still alive, two-thirds of them died within 10 years. Two patients were alive more than 15 years after the operation. In some cases the patient has had some troubles: certain stiffness of the peripheral joints (fingers and toes), sensations of numbness, coldness, or tiredness, and occasionally pains after exertion. These symptoms indicate that the circulation has not been fully restored, and might in some cases depend upon small embolisms in the peripheral vessels.

SPASMOPHILIC DRUGS

An embolus often produces a spasm in the part of the wall of the vessel where it lodges and thus disturbs the circulation still further. For this reason Peiser has suggested the use of a spasmolytic substance which would do away with the vascular cramp and improve the circulation. Thus intravenous injections of Eupaverin have been tried. Denk has recently reported the use of this treatment on 5 of his own cases of emboli in the extremities and 20 others from the literature, as well as 9 cases of pulmonary emboli. The result must be regarded as remarkable, for in the former group 17 were cured, 3 improved, and 5 unimproved, while in the latter group 7 survived and 2 died. It would seem to be too early to give a final judgement on this method of treatment, but the results are remarkably good. In my opinion one should not neglect its use; but even if such intravenous injections can improve the circulation when an embolus is producing grave circulatory disturbance in one of the extremities, I consider that the embolus should be removed; otherwise one may be forced to do this later, when the prospect of a good result is much poorer. We shall indeed have gained a great deal if this method decreases the mortality from large massive embolism in the pulmonary artery.

CONCLUSION

An embolus which causes circulatory disturbance of a threatening character in the upper or lower extremities ought to be removed by arteriotomy unless there are contra-indications present. Moreover, the operation ought to take place as soon as possible. The sites chiefly involved in such cases have already been mentioned.

As the embolus suitable for operation is far from rare, it is of importance that practising medical men should learn to diagnose it in time, and that they should immediately consult a surgeon or send the case to the surgical department of a hospital.

BILATERAL LOBECTOMY FOR BRONCHIECTASIS

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THE removal of lungs or lobes of lung with any consistent success is so recent that the operative criteria have scarcely taken form, let alone finality. Most thoracic surgeons have been inclined to the view that in bronchiectasis the disease should be confined to a single lobe, which is usually a lower lobe, if surgical treatment is to be undertaken. Or, in the case of the right lung, the middle lobe also, if affected, might be removed with the lower. Still more recently a number of surgeons in different parts of the world have reported 17 cases of total pneumonectomy for infective disease involving the whole of a lung. Cases of bilateral bronchiectasis involving both lower lobes, however, have been considered to be definitely beyond the scope of surgery, and as far as the writer knows the only successful case of the kind that has been described is that of Eloesser. Alexander also refers to four cases of his where this procedure was proposed, but it is not quite clear how many survived. There is no record of any in this country. Previous attempts at bilateral lobectomy have usually not been carried to a successful issue because the patient is so ill after the first operation; this seems to make the opposite diseased side flare up in a remarkable way. It appears, therefore, that an account of a successful case might be of some interest in a field of surgery where both the principles and technique are in the formative stage.

The remarkable advances in the surgery of the lung during the last five or six years have been made possible only by the development of radiography to its present high pitch, and particularly the introduction of bronchography. A decade before, the introduction of opaque media had revolutionized the diagnosis of diseases of the alimentary canal; so, in the twenties, a similar development in the case of the respiratory tract added certainty and precision to the diagnosis of pulmonary disease. In each case there followed a remarkable sharpening of interest and extension in surgical treatment.

Regarding the alimentary tract, it is already being realized that enthusiasm sometimes, as in the case of peptic ulcer, rather outstripped judgement. It is possible that the same is now happening in the case of diseases of the lung. But it will be many years before it is possible to assess the ultimate value of lobectomy, let alone pneumonectomy.

SPECIAL CONSIDERATIONS

Radiography.—A careful lipiodol radiograph is an essential pre-requisite to any surgical treatment. The exact topography of the disease must be ascertained. In the bilateral case here recorded, the first bronchography showed that both lower lobes were affected. Subsequent bronchographies were then carried out at intervals of a week, doing one lung at a time so as to secure filling of the upper and middle

main bronchioles in particular. In this way it is possible to obtain complete and unconfusing pictures of right and left bronchial trees separately.

Vital Capacity.—The considerations which apply to the bilateral case differ in several respects from those applying to solitary lobectomy or even to total pneumonectomy. The cardinal fact in the latter is that the patient is in possession of at any rate one healthy lung. The success of the operation depends on the adequacy of that lung, not only during the operation, but during those anxious days following. Its serious involvement, as by aspiration broncho-pneumonia or contralateral pneumothorax, is likely to be fatal. The bilateral case has to rely for a time, after the first lobectomy, on a lung part of which is diseased. Moreover, during and after the second lobectomy the patient has to rely on the recovered upper lobe of the lung first operated on. Problems that arise are thus the vital capacity of the particular patient with (a) one lung, (b) one lobe, working, and also the very difficult problem of the most suitable anæsthetic.

Fortunately the adequacy of the vital capacity under conditions (a) and (b) can be determined with fair certainty pre-operatively from the patient's condition under preliminary pneumothorax. In the bilateral case, with this end in view, a pre-operative pneumothorax down to less than half the lung volume is probably an advantage.

Anæsthesia.—Much thought and ingenuity has been expended by anæsthetists and surgeons on the peculiar problems of anæsthesia for major thoracotomies. The progress from negative-pressure chamber to positive-pressure endotracheal anæsthesia, to closed mask, to spinal anæsthesia, has corresponded with the increased knowledge of the physiologist, and even more with the increased confidence as the result of experience of the surgeon. An endotracheal catheter by its trauma undoubtedly increases the liability to tracheobronchitis. Positive-pressure anæsthesia must tend to distribute pus and any particulate matter throughout the lungs. In unilateral disease the ingenious endobronchial catheters with inflatable cuffs may minimize this danger, but obviously cannot help in the bilateral case. Here the ideal seems to be a spinal anæsthetic.

The practice of inflating the remaining lobe by positive pressure when closing the chest seems to me to be not without danger, as this must force infected material to the terminal bronchioles or even infundibula. A much safer method to secure early expansion is to empty the pleural cavity of most of its air by means of the artificial pneumothorax apparatus before the patient leaves the operating table.

Whether bilateral lobectomy for bronchiectasis is a sound procedure from a pathological point of view must remain a matter for suspended judgement for the present, as for that matter must solitary lobectomy and pneumonectomy. Bronchiectasis is, of course, a progressive disease. Whether it is also a necessarily *spreading* disease is not clear. It is known that in the neighbourhood of an abscess of the lung the bronchioles after a time become bronchiectatic. It is therefore all too probable that healthy bronchioles adjacent to a bronchiectasis likewise gradually become involved by the disease. Arrest of the spread can probably be secured by the removal of the stagnant focus of infection.

The Stage of the Disease Suitable for Operation.—Here again the considerations are rather different from the unilobar case. Accepting the rough descriptive stages of dry, septic, and putrid bronchiectasis, it may be said that the first is too early and the last too late, as long as the operative hazards remain what

they are. In the second group there is probably a considerable number of bilateral cases that can be successfully treated by surgery. The main problem is to recognize them before they reach the putrid stage and become too toxic and emaciated. The most certain way would seem to be to regard patients, especially children, with chronic bronchitis, bronchiolitis, or recurrent attacks of bronchopneumonia, as probably bronchiectasis and to investigate the matter by bronchography. The suspicion is particularly likely to be confirmed if there is clubbing of the fingers.

CASE REPORT

I. R., a tall, fair-haired girl of seventeen, was admitted to the North Middlesex Hospital on Dec. 2, 1935, suffering from an exacerbation of a chronic respiratory infection. She had a cough, pain in the chest, shortness of breath, a slight temperature, and copious yellow sputum, dullness of the left base, with bronchial breathing. This attack soon settled down to a chronic state of cough, expectoration 2 to 2½ oz. of pus daily—not offensive—and a temperature occasionally rising to 99° but with a pulse round about 90. She had moderate clubbing of the fingers. There were always extensive râles over both bases. Weight, 7 stone 2 lb. on admission.



FIG. 246.—Bilateral bronchiectasis. Bronchograph taken December, 1935, before operation.

PREVIOUS HISTORY.—There was a chest illness at the age of 9 years, for which she was three months in another hospital. Since that time she had spent most of her life either in hospital, or in a convalescent home in the Isle of Wight “getting over the chest trouble”.

INVESTIGATIONS.—Radiography of the chest showed some fibrosis at the bases. *Lipiodol* showed marked bronchiectasis of both lower lobes, cylindrical on both sides, but with saccular areas as well in the right base (Fig. 246). Both lungs were later done separately, and as far as could be judged the condition was confined to the lower lobes.

In the meantime steps were taken to improve the patient's condition. Postural drainage was secured on a Nelson bed, plenty of nourishing food was given, and daily actinotherapy. By the end of March she was much stronger and had put on a stone in weight. It was then thought that the maximum improvement had probably been obtained, and it was decided to attempt ablation of the lower lobes one at a time. She still brought up 2 to 3 oz. of pus a day. The left was clinically the worse side and so was done first.

Left artificial pneumothorax was carried out, 1400 c.c. of air being introduced within the ten days preceding operation. Extensive basal adhesions could be seen on the X-ray film.

FIRST OPERATION: LEFT LOWER LOBE. April 17, 1936.—Anæsthetic administered by Dr. John Challis—12.5 c.c. of spinal percaïne supplemented with a little gas and oxygen with face-piece. During the spinal puncture and throughout the operation the patient was carefully kept on her 'good' side with the thoracic spine arched upwards and the head low. The anæsthesia on the operated side reached the clavicle, while the under side was little affected as judged by the respiration.

A left postero-lateral incision was made in the 6th intercostal space. A quarter of an inch of the 6th rib behind the angle was resected. Extensive pleural adhesions were present over the lower lobe, more especially to the diaphragm. These were slowly divided, mostly by blunt dissection. The pedicle was controlled by Roberts' tourniquet, and divided rather shorter than usual. The stump was swabbed with N.A.B. Forty-day No. 2 catgut was used for multiple mattress sutures to occlude vessels and bronchioles. The pleura was over-sutured, but not very completely. The chest was then closed: pericostal catguts; two layers of muscle catguts; skin—six s.w.g. and fine continuous silk. In the tenth intercostal space a tube was led to a water-seal drainage system. The operation lasted 1½ hours.

The condition of the patient after the operation was rather bad for about twelve hours. She was pale, with a pulse of 120 and of poor volume. After that she steadily improved. The tube had ceased draining by the third day. On the third morning the small remaining pneumothorax (500 c.c.) was withdrawn with artificial pneumothorax apparatus through the first intercostal space anteriorly. There was very slight surgical emphysema within three inches of the wound.

On the fourth and fifth day there was a transient increase of infection of the right lower lobe, with a small area of bronchial breathing and expectoration. The wound healed by first intention, and the tube track remained dry after the tube was removed on the 13th day.

May 7: Patient was getting up and walking at the end of three weeks and resuming actinotherapy and exercise. Expectoration is now reduced to 1½ oz. a day including postural drainage.

June 2: Patient now very fit, artificial pneumothorax on second side induced this day after two previous unsuccessful attempts due to extensive adhesions. Altogether 2000 c.c. air introduced in three amounts. Lung collapsed to about one-third. At the base, owing to extensive adhesions, moist sounds can still be heard, while the rest of the lung is silent.

SECOND OPERATION: RIGHT LOWER LOBE. June 11.—The anæsthetist again was Dr. John Challis—12 c.c. of percaïne were introduced in the third lumbar space. At times during operation a little oxygen was given on a mask, more as a placebo than on account of any respiratory distress.

A long postero-lateral intercostal incision was made. Bleeding points were sealed with diathermy. Rib not resected; exposure quite good enough. Very extensive and thick adhesions were present over the whole lower lobe, also a wide one over middle of upper lobe. These were divided, the worst ones with diathermy. The phrenic adhesions were virtually continuous, and the lung had to be laboriously peeled off. A fair amount of bleeding took place from a vessel in the diaphragm, which was ligated with a suture. Ligamentum latum clamped and divided. Adhesions were even worse in the interlobar fissure, and it was only with the greatest difficulty that it could be identified and separated at all. Eventually it was separated without any apparent hole into the parenchyma. Roberts' tourniquet was applied to the pedicle, and two-way No. 2 catgut mattress sutures used. Pleural inversion followed with No. 1 catgut. N.A.B. was used on the raw pedicle. No positive-pressure inflation was used at the end. The chest wall was closed—airtight—with pericostal catguts and continuous suture for muscles; for the skin, continuous fine silk plus four s.w.g. was used. An intercostal tube was placed laterally through the tenth space. Before the patient left the table 2000 c.c. of air was aspirated from the pleura with artificial pneumothorax apparatus. The operation lasted 1 hour 20 minutes.

The patient's condition was remarkably good throughout. She was a little ashen but never cyanosed, and the pulse at the end of the operation was only 116 and of quite good volume.

June 13: Colour now quite pink. No distress. Radiograph shows almost full expansion. Breath-sounds coming through well. Other side: no adventitious sounds. Water-level oscillation ceased this morning (40 hours after operation) with the coming together of

the pleural surfaces. Only a small fluid opacity in costo-phrenic angle. Total drainage of bloody fluid since operation—one pint.

June 14: Upper lobe fills hemithorax. Some moist sounds under right breast, and patient is coughing up a few drachms of thin purulent sputum.

June 17: For last two days patient has had intense pain in abdomen and has been awake all night. She refuses to lie down as it makes pain worse. Maximum pain is one inch below umbilicus to the right. The intercostal tube was removed. (It was catheter size No. 16 and fitted very tightly.)

June 18: No pain at all since removal of tube. General condition has been excellent.

July 12: Recovery uneventful except for the intercostal nerve pain. There was never any real anxiety over her condition after the second lobectomy. There was a mild infection of the tube-track only (temperature never above 100°). She got up on the nineteenth day. The wound healed by first intention. There was no surgical emphysema on this occasion. Only 1 drachm expectoration now, and this is mucoid. No appreciable pus. Quite strong,



FIG. 247.—Bronchograph taken July, 1936, after bilateral lobectomy.



FIG. 248.—Showing position of scars after bilateral lobectomy.

and takes exercise in the grounds freely without any shortness of breath. Condition on radiography with lipiodol is shown in *Fig. 247*, and the position of the incisions in *Fig. 248*.

Sept. 5: For the last six weeks there has been no phlegm. She is now quite strong and is working as a ward maid.

PATHOLOGY.—Each of the removed lobes was greatly contracted and covered with a thickened pleura which had been adherent to the chest wall. The fresh specimens had lost the pink, aerated look of normal lung, and were of a dark fleshy appearance, although there were considerable surface areas still of normal colour. The texture was spongy in these parts, but elsewhere solid fibrous areas could be felt. The bronchioles were practically all dilated and stood out from the surrounding cut surface; pus could be squeezed from them. A few saccules of pus the size of currants were found, but mostly the bronchiectasis was cylindrical. The lung parenchyma was fibrotic around the bronchial tubes. There were also tracts of round-celled infiltration and fibrosis extending between neighbouring tubes. The mucous membrane was much thickened and the mucous glands hyperplastic.

SOME POINTS IN TECHNIQUE

The success of the operations was due, I think, to attention to the following points in the technique.

Great pains were taken to improve the patient's general state before the operation by postural drainage, good food, and actinotherapy. She gained a stone in weight.

As for surgical technique, there is little that is different from the unilateral case. The Brunn-Shenstone one-stage operation appears to be steadily gaining ground on the two-stage 'strangling' operations. It commends itself as a cleaner surgical proposition, and the mortality of some of the best series, such as that of Tudor Edwards and Thomas, compares favourably with the best two-stage mortality, e.g., Alexander's. The reasons in favour of preliminary artificial pneumothorax are more cogent than even in the unilateral case.

In each lobe the pedicle was dealt with by the multiple mattress suture method described by Tudor Edwards. The pleural over-suture, while snug, had no pretence to being air-tight in either case, and the security of the pedicle seems to depend but little on this. No fistula occurred on either side. The rib above the incision was resected at the angle in the first lobectomy, but not in the second. In both cases the approach was entirely adequate, but in the second there was no surgical emphysema whatever of the chest wall, owing, no doubt, to the better apposition obtained with the rib intact. If air passes into the wound so may infection from the pedicle. I have on other occasions observed that better apposition of the wound can be obtained if no ribs are divided.

On both sides water-seal drainage through a low intercostal tube was employed. As usual the tube drained for only about forty-eight hours. A 'portable' radiograph of the chest was taken daily for six days after each operation. The blood was grouped in readiness, but no transfusion was needed.

The thoracic wounds were covered merely by a strip of gauze held in place with short lengths of strapping. There is in this way no interference with respiratory movements after the operation. The patient was alternately sitting up and lying on one or other side, so as to lessen the chances of hypostatic pneumonia.

SUMMARY

1. In addition to the two cases previously described in the literature, a case of successful bilateral lobectomy for bronchiectasis is described.

2. The limited indications for such a procedure and the special considerations in technique and management are discussed.

3. It is suggested that in the bilateral case reasonably early diagnosis is specially important if surgery is to be of help, and all cases of chronic or recurrent affections of the lungs, particularly in adolescents, must be regarded as possible bronchiectases.

BIBLIOGRAPHY

- ELOESSER, L., "Bilateral Lobectomy", *Surg. Gynecol. and Obst.*, 1933, lvii, 247.
ALEXANDER, J., "Observations on Total Pulmonary Lobectomy and Pneumonectomy," *Ann. of Surg.*, 1935, ci, 393.

EXPERIMENTAL SURGERY

NERVE GRAFTING

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INTRODUCTION

IT has been shown several times during the last twenty-five years, by experimental work in animals, that a nerve-graft will serve to bridge successfully a nerve gap, so that, in time, the anatomical and physiological continuity of the nerve is restored. When, however, at the time of the Great War, nerve-grafts were used to repair peripheral-nerve defects in man, the results were far from satisfactory. A full résumé of the literature up to 1934 is given by Stookey¹ and by Davis and Cleveland,² and need not be repeated here. The main feature which emerges from a perusal of this voluminous literature is the great diversity of opinion that exists on the value of a nerve-graft in peripheral-nerve surgery.

French surgeons^{3, 4, 5} believe that a successful result can be obtained, and some have gone so far as to use grafts from cadavera and from animals, first storing the grafts in alcohol in order to kill the living cellular elements, and have claimed successful results with such grafts. The published case records, however, are brief, and are unsatisfactory inasmuch as late results are not given, and so many contradictory results are recorded that it is not possible to form an exact opinion on the value of their procedures.

English and American surgeons found their results were on the whole poor: Stopford⁶ recorded 20 cases of autogenous nerve-grafts, in none of which was there evidence of recovery of function. Platt⁷ expressed more or less the same opinion as a result of his experiences with 15 cases of nerve-graft plus fascial tubalization, and Platt and Bristow⁸ in a comprehensive survey of the subject found the results so disappointing that they concluded: "The attitude of British surgeons towards the value of the nerve-graft continues to be one of scepticism."

The nerve-graft, therefore, remained condemned until the work of Duel and Ballance in 1931.^{9, 10} These workers showed, first in monkeys and then in man, that recovery of function followed the implantation of a short nerve-graft into a defect in the petrous portion of the facial nerve. The protocols of their experiments, and the films and pictures of their results, show conclusively that under the conditions of their work, recovery of function followed the implantation of a nerve-graft.

Duel and Ballance, however, were led further by a study of the writings of Cajal. Cajal had accepted an observation of Tello,¹¹ that portions of fresh nerve implanted into the cerebral substance were absorbed, whilst pieces of degenerated nerve attracted the nerve-fibres of the brain. Tello's conclusion was that "the empty channels of the degenerated grafts are especially rich in neurotropic

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substances, whereas in the normal nerve grafted directly, though not entirely absent, they are liberated very tardily". Cajal had an equal belief in the neurotropic attraction in a piece of degenerating nerve. He writes¹²: "The ideal (nerve) graft is the peripheral stump newly taken from an operated animal 8 to 15 days after the operation. The newly formed fibres travel through the empty sheaths with extraordinary speed, deviations and retrogressions being much diminished." Ducl and Ballance, therefore, started to insert pieces of degenerated nerve as grafts, and claimed that in such cases recovery of function was obtained in a quarter to half the time taken following the use of fresh grafts.^{13, 14, 15} This advantage of a degenerated graft over a fresh one was expressed in no uncertain terms, and Ballance gave three reasons for the advantage:—

1. The products of Wallerian degeneration within the degenerated graft exert a neurotropic attraction on the down-growing nerve-fibres (Cajal and Tello).

2. As the undisturbed peripheral end of a divided nerve has an intact blood-supply, the products of Wallerian degeneration are rapidly removed, leaving the neurilemmal tubes empty. If such a piece of nerve is used as a graft, these tubes are ready to receive new nerve-fibres immediately. Whereas a piece of fresh nerve used as a graft obtains a new blood-supply only slowly, the products of Wallerian degeneration are therefore not removed for some time, and form a barrier to the down-growth of the new fibres.

3. During the time taken for the vascularization of the fresh nerve-graft and the clearing of the neurilemmal tubes, the fresh graft is "a foreign body, which becomes surrounded in two weeks by young fibrous tissue which blocks the ends of the graft, thus forming a barrier to the immediate entrance of axis cylinders: much time is required before the axons are able to pierce this cork of young fibrous tissue, whereas the axis cylinders can and do enter at once the freshened ends of a degenerated graft".

It should be noted that there are three conditions implied in this argument:—

1. That the products of Wallerian degeneration do exert a neurotropic attraction on new-growing nerve-fibres.

2. That the old neurilemmal tubes do persist and allow new nerve-fibres to traverse them, and that the presence of the lipoidal masses—products of Wallerian degeneration—form a barrier to the down-growth of these fibres.

3. That the fresh nerve-graft provokes a foreign-body reaction whilst the degenerated nerve-graft does not.

EXPERIMENTS

In collaboration with Sir Charles Ballance, experiments were begun at the Royal College of Surgeons eighteen months ago, to investigate more fully the use and principle of the degenerated nerve-graft, and to try to determine the reason for the poor results of nerve-grafting in peripheral-nerve lesions in man.

METHODS

1. **Nerve-suture Experiments.**—These experiments were carried out to determine whether the rate of recovery of function was influenced in any way by the state of degeneration in the peripheral-nerve segment.

The external popliteal nerve of the cat was used throughout the investigation, since the resulting paralysis troubles the cat little and there is no tendency to

trophic ulceration. Under ether anæsthesia the nerve was exposed in the right thigh, and divided at the level of the upper border of the gastrocnemius. Nine to twenty-one days later the nerve-ends were re-exposed, freshened, and sutured. At this second operation the external popliteal nerve of the left leg was divided at the same level and immediately sutured. Fine silk on a fine atraumatic needle was employed for suture material and great care was taken to obtain accurate end-to-end approximation of the nerve, and to pass the sutures, two at each suture line, through the nerve-sheath only. A certain number of days after this operation, under chloralose anæsthesia, the nerves were exposed on each side proximal to the suture lines and stimulated electrically. The presence or absence of movements of the exposed peronei and extensor longus digitorum tendons as a result of this stimulation was noted, and the cat then killed.

To avoid aberrant results from spread of current we have not used the faradic coil as the source of stimulation, but the discharge from a condenser of small capacity. The condensers used have been from 0.002 to 0.006 microfarads, charged with a 2-volt battery, and the charge, therefore, thrown into the nerve is so very small that aberrant results from spread of current are eliminated.

2. Nerve-graft Experiments.—

a. AUTO-GRAFT.—The external popliteal nerve of the cat was again employed as the subject of the experiments, but at a higher level in the thigh than in the nerve-suture experiments in order to obtain a more circular compact piece of nerve than the rather flattened ribbon available below. The graft was taken, under ether anæsthesia, from the external popliteal nerve in the thigh, and implanted in a corresponding defect in the upper third of the external popliteal nerve of the other leg. In some experiments a piece of normal (fresh) nerve was used as a graft; in other experiments the donor external popliteal nerve was first divided and allowed to degenerate *in situ*, and grafts of different numbers of days' degeneration were used. The site of implantation of the graft was kept as constant as possible, as well as the length of the graft—3 cm., which represents about one quarter of the total length of the external popliteal nerve in the thigh. We placed great importance on the technique and accuracy of suturing and on exact correspondence between the size of the cut end of the graft and of the recipient nerve.

A certain number of days after operation, under chloralose anæsthesia, the nerve was exposed proximal to the graft and stimulated electrically as in the nerve-suture experiments. Presence or absence of movements of the exposed tendons of the peronei and extensor longus digitorum as a result of stimulation was noted; the whole length of the nerve was then excised from above the graft down to the level of the ankle, and the cat killed. The length of nerve was pinned out on cork, and fixed in one piece in ammonia-alcohol.

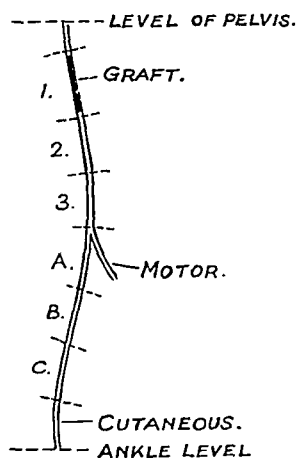


FIG. 249.—Diagram of nerve segments.

When fixation was complete, the nerve was taken and divided into segments as follows: The site of origin of the motor branch was the fixed point from which

measurements were made: from this point lengths of nerve 3.5 cm. in length were cut off (*Fig. 249*). These segments of nerve were stained by Ranson's silver impregnation method, and serially sectioned. In this way we had a record of the extent of regeneration along the main trunk of the nerve and its long sensory branch, and a measurement of the distance of the graft from the motor branch—an important point in making comparisons of times of recovery of motor function.

b. HOMEO-GRAFT.—The procedure was the same as in the autogenous nerve-graft experiments, except that the graft was taken from one cat and implanted into a second one. The procedure differed also from the auto-graft experiments in that both external popliteal nerves were grafted, a fresh graft being implanted into the left nerve and a degenerated graft into the right.

RESULTS

1. Nerve-suture Experiments.—In each animal two parallel experiments had been carried out. On the one side (the right) the nerve had been divided and sutured only after a period of degeneration varying from nine to nineteen days. On the other side the nerve had been divided and sutured immediately.

Table I shows the result of the full series of this experiment. It will be seen that 42 or more days after operation (Cats 1-7) motor function was equally present on the two sides; that after 35 days (Cat 8) motor recovery had occurred on both sides, although on the right—previously degenerated—side both extensor longus digitorum and the peronei contracted, while on the left contraction of the extensor only was observed; and that 28 days or less after operation (Cats 9-11) recovery of motor function had not occurred on either side.

Table I.—RESULTS OF NERVE-SUTURE EXPERIMENTS

No OF CAT	NUMBER OF DAYS' DEGENERATION OF RIGHT NERVE BEFORE SUTURE	NUMBER OF DAYS BETWEEN NERVE-SUTURE OPERATIONS AND SUBSEQUENT EXPOSURE AND STIMULATION	MOVEMENTS OF EXTENSOR AND PERONEI TENDONS ON NERVE STIMULATION	
			Right Side (preliminary degeneration)	Left Side (immediate suture)
1	9	112	+	—
2	11	103	—	—
3	13	89	complete absence of regeneration	—
4	9	76	—	—
5	14	62	—	—
6	10	53	+	—
7	15	42	—	—
8	13	35	—	—
9	14	28	—	—
10	12	22	—	—
11	19	15	—	—

* Only extensor longus digitorum No peronei response.

The difference between the regenerative processes on the two sides was therefore very slight, and not sufficient to suggest any effective increase in neurotropic attraction in the previously degenerated nerve. One would not therefore expect any difference in neurotropic attraction between a fresh and a degenerated nerve-graft, unless the onset of Wallerian degeneration is delayed in an isolated piece of

nerve (fresh graft) or the products of this degeneration in the isolated nerve differ in some way from those in a nerve degenerating in situ. We have evidence, however, to show that Wallerian degeneration is in thirteen days as advanced in the one as in the other.

Experiment.—The right external popliteal nerve of a cat was divided high up in the thigh, taking care not to strip the nerve from its bed. At the same operation 3 cm. of the left external popliteal nerve in the thigh was excised and laid in an intermuscular plane. Thirteen days later a portion of the degenerated nerve from the right side and the isolated piece of nerve from the left side were removed, fixed, and stained by Ranson's method, and sectioned. In the piece of nerve degenerated in situ and in the isolated piece of nerve, Wallerian degeneration had reached an advanced stage (*Figs. 250, 251*). In both, the axis cylinders were fragmented and the thickened neurilemmal tubes filled with lipoidal masses. The



FIG 250—Longitudinal section of peripheral end of nerve divided in situ, after 13 days. Note extent of Wallerian degeneration, and compare with *Fig 251* ($\times 135$)

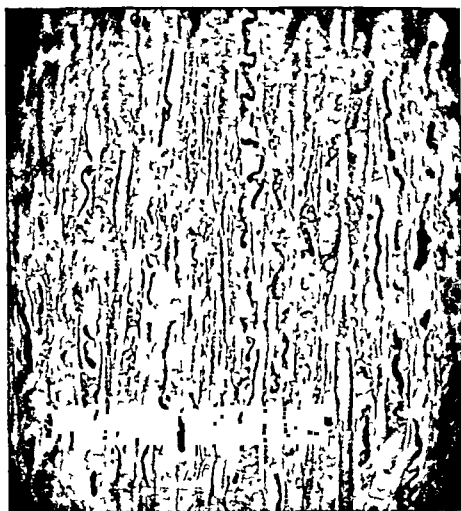


FIG 251—Longitudinal section of isolated piece of nerve, after 13 days. Note extent of Wallerian degeneration, and compare with *Fig 250* ($\times 135$)

appearances in the two are quite comparable except that absorption of the products of Wallerian degeneration is more advanced in the piece of nerve degenerated in situ (and therefore with an intact blood-supply) than in the isolated piece of nerve. Vascularization of the isolated piece of nerve, however, has begun, and capillaries have already penetrated into the outer parts of the nerve, so that absorption of the products of Wallerian degeneration is occurring in those parts. It does not appear, therefore, that after 13 days the state of Wallerian degeneration differs very much in an isolated piece of nerve and in a nerve degenerated in situ.

We have concluded from these experiments that preliminary degeneration does not in itself facilitate subsequent axonal growth, and that any advantage from possible neurotropic attraction in the degenerated graft can be disregarded. It should be mentioned here that there is in addition evidence from the work of

Weiss¹⁶ that neurotropic attraction plays no part in the growth of nerve axons, and that the factors responsible for directing growing axons along a nerve are physical and not chemical.

If, then, any superiority of the degenerated graft exists, it must depend on the two other factors described by Duel and Ballance—namely, the blocking of the fresh nerve-graft by unabsorbed products of Wallerian degeneration or by the connective-tissue reaction that the fresh graft provokes.

2. Nerve-graft Experiments.—

a. AUTO-GRAFT.—Fresh nerve-grafts had been inserted in 5 cats and degenerated grafts in a similar number. Since the upper end of the graft was about 6 cm. proximal to the site of division and suture in the nerve-suture experiments, we did not anticipate recovery of motor function in the grafting experiments for some twenty to thirty days more than the 35 days taken after nerve-suture. Accordingly we did not commence to stimulate the nerve until 57 days after operation. As motor recovery was found in some instances at that time it may be that the earliest stages of recovery were missed, for an experiment that showed such a recovery on the 57th day might have shown recovery a few days earlier. The purpose of the serial sections of the whole length of nerve, however, was to provide a record of the extent of regeneration, and in comparing the results of fresh and degenerated nerve-grafts we have based our opinion on the extent of nerve regeneration shown in the preparations, as well as on the results of stimulation.

The distance from the proximal suture line to the farthest point that new axonal growth could be traced was measured in each case. The figure is not accurate to more than 1 cm. or so, since the thin terminal portions of the nerve stained unequally, and it was difficult at times to be certain whether nerve-fibres were present in the end segment or not. There can be little significance, however, in a small difference in the extent of nerve-axon growth in two cases, since variations in rate of growth and in accuracy of approximation and in scar-tissue formation at the suture lines will readily cause differences in the regenerative processes, and it is only a large difference in these processes, such as the one described by Duel and Ballance, that can be of significance in a comparison of this kind.

Table II gives the results of the experiments. It will be seen that *Cat 1* (58-day fresh-graft experiment) is to be compared with *Cat 6* (57-day degenerated-graft experiment). It appeared at first that here was an advantage of the degenerated graft over the fresh, since there was recovery of motor function in the former and not in the latter; but measurement showed that the degenerated graft had been inserted 0.5 cm. more peripherally than the fresh, and that the actual distance the axons had grown in the two cases was the same (15.5 and 16.0 cm.).

Cat 2 (61-day fresh-graft experiment) and *Cat 8* (59-day degenerated-graft experiment) are comparable. In the fresh-graft experiment there was a small muscular contraction, indicating that relatively few axons had reached the muscle, and new nerve-fibres could be traced 15.5 cm. along the nerve. In the degenerated-graft experiment there was also a small muscular contraction, and the distance of new fibre growth was 17.0 cm. The grafts in these two experiments had been inserted at a similar height in the thigh (10 cm. above the motor branch in each case), and the comparison is therefore just.

Table II.—RESULTS OF AUTO-GRAFT EXPERIMENTS

NO. OF CAT	NUMBER OF DAYS DEGENERATION OF GRAFT	NUMBER OF DAYS BETWEEN OPERATION AND SUBSEQUENT EXPOSURE AND STIMULATION	MOVEMENTS OF EXTENSOR TENDONS AS RESULT OF NERVE STIMULATION	DISTANCE FROM PROXIMAL END OF GRAFT TO MOTOR BRANCH	TOTAL DISTANCE NEW AXONS HAVE GROWN
Fresh-nerve Graft				cm.	cm.
1	—	58	—	10.5	16.0
2	—	61	+	10.0	15.5
			(very slight)		
3	—	64	+	8.8	17.5
4	—	73	+	8.5	17.5
5	—	88	+	9.0	18.5
Degenerated-nerve Graft					
6	15	57	+	10.0	15.5
7	17	58	died before stimulation	10.5	14.0
8	17	59	+	10.0	17.0
			(very slight)		
9	14	60	—	10.5	16.0 (but very few fibres)
10	14	73	+	8.8	17.0

Cat 3 (64-day fresh-graft experiment) is to be compared with *Cat 9* (60-day degenerated-graft experiment). Motor recovery had occurred in the former but not in the latter, but it must be noted that the fresh graft was situated 1.7 cm. more peripherally than the degenerated graft. Nerve-axons had, however, grown a greater distance in the fresh-graft experiment than in the degenerated one.

Thus in the three fresh-graft experiments stimulated about the sixtieth day after operation (*Cats 1, 2, and 3*), there was recovery of motor function in two, and the average distance the nerve-fibres had grown was about 16 cm. In the three corresponding degenerated-graft cases there was recovery of motor function in two, and the average length of new axonal growth was also about 16 cm.

At 73 days motor activity was present in both types of experiment (*Cats 4 and 10*), and in each nerve-fibres could be traced about 17 cm.

We have thus not found any obvious difference in the time taken for recovery of motor function or in the distance that the new axons have grown in a given time between the fresh- and degenerated-nerve-graft experiments. It was possible, moreover, to obtain further comparison from the sections of the grafts and nerves at different levels.

At the 60-day period both types of grafts were packed with nerve fibres, and in no case was there any obvious diminution in the number of fibres in one graft compared with the others (*Figs. 252, 253*). The age of the fibres as judged histologically was also much the same. In nerve-regeneration preparations the older of the new axons are thick and stain brown, while the young, more recently grown, fibres are finer and stain dark black. In the 6 experiments examined around the 60-day period the proportion of thick and fine fibres was very comparable in the two types of graft, and it was evident that a preponderance of the thicker, older fibres did not belong to either fresh or degenerated grafts (*Figs. 254, 255*).

The number of fibres seen in segments of nerve remote from the graft (e.g., segment 3, or A, in *Fig. 249*) also afforded a good basis of comparison of the extent



FIG. 252.—Longitudinal section of fresh autograft, 58 days after operation (*Car 1*). Full width of graft. Note presence of nerve-fibres, and compare with *Fig. 253*. ($\times 28$)



FIG. 253.—Longitudinal section of degenerated autograft, 59 days after operation (*Car 8*). Full width of graft. Note presence of nerve-fibres, and compare with *Fig. 252*. ($\times 28$)



FIG. 254.—Higher magnification of graft shown in *Fig. 252*. Note proportion of thick and fine fibres, and compare with *Fig. 255*. ($\times 135$)



FIG. 255.—Higher magnification of graft shown in *Fig. 253*. Note proportion of thick and fine fibres, and compare with *Fig. 254*. ($\times 135$)

of regeneration, and on this basis the extent of regeneration in the two series of experiments was again found to be indistinguishable. Where equal accuracy of

approximation had been obtained at operation very similar numbers of fibres were observed in the nerves at equal distances below fresh and degenerated grafts (*Figs. 256, 257*).

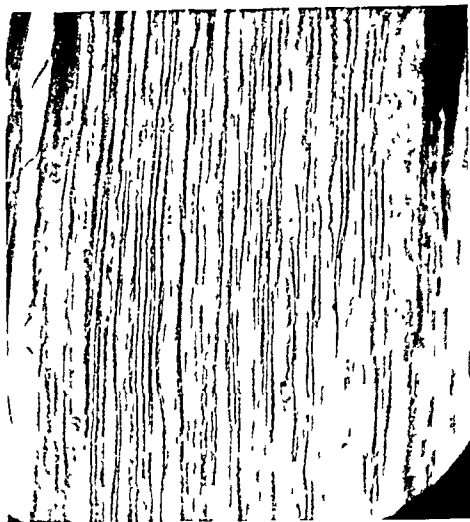


FIG 256.—Longitudinal section of nerve 6 cm. distal to fresh auto-graft, 58 days after operation (*Cat 1*). Full width of nerve. Note presence of nerve-fibres, and compare with *Fig. 257*. ($\times 135$)



FIG. 257.—Longitudinal section of nerve 6 cm. distal to degenerated auto-graft, 59 days after operation (*Cat 8*). Full width of nerve. Note presence of nerve-fibres, and compare with *Fig. 256* ($\times 135$)



FIG 258.—Longitudinal section of peripheral end of nerve divided in situ, after 13 days. Note new nerve-fibres lying between old neurilemmal tubes. ($\times 320$.)

Observations on the histological preparations therefore confirmed the similarity of the regenerative processes in the two types of experiment.

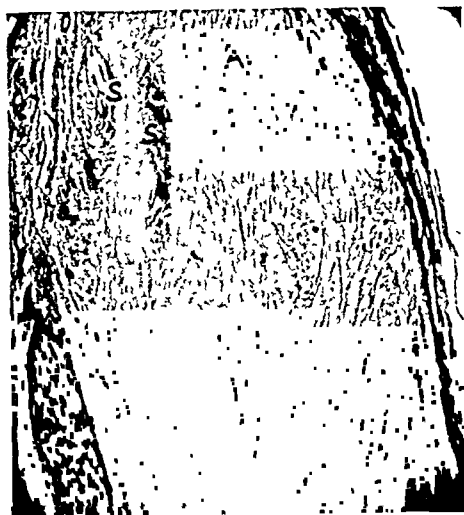


FIG. 259—Longitudinal section of proximal suture line of fresh auto-graft experiment, 58 days after operation (*Cat 1*). Full width of anastomosis. Note amount of scar producing deviations of the nerve-fibres, and compare with *Figs 260, 261, and 262*. A, Proximal nerve entering the anastomosis. S, Suture, marginally placed. ($\times 28$)



FIG. 260—Longitudinal section of proximal suture line of fresh auto-graft experiment, 64 days after operation (*Cat 3*). Full width of anastomosis. Note amount of scar producing deviations of the nerve-fibres, and compare with *Figs. 259, 261, and 262*. A, Proximal nerve entering the anastomosis, S, Suture, marginally placed. ($\times 28$)



FIG. 261—Longitudinal section of proximal suture line of degenerated auto-graft experiment, 60 days after operation (*Cat 9*). Full width of anastomosis. Note amount of scar producing deviations of the nerve-fibres, and compare with *Figs. 259, 260, and 262*. A, Proximal nerve entering the anastomosis. ($\times 28$)

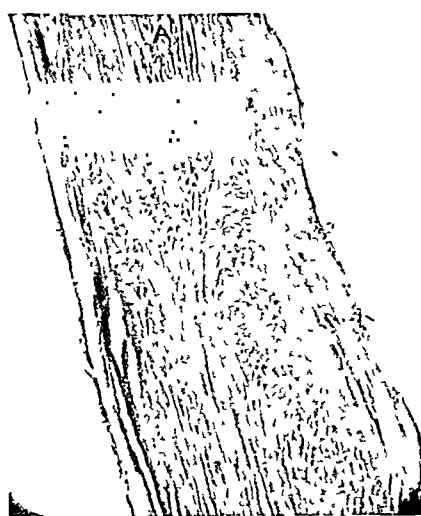


FIG. 262.—Longitudinal section of proximal suture line of degenerated auto-graft experiment, 57 days after operation (*Cat 6*). Full width of anastomosis. Note amount of scar producing deviations of the nerve-fibres, and compare with *Figs. 259, 260, and 261*. A, Proximal nerve entering the anastomosis. ($\times 28$)

It is of interest to consider here the two remaining theoretical conceptions on which Duel and Ballance had explained their results.

Obstruction of the Neurilemmal Tubes of the Fresh Graft by Unabsorbed Products of Wallerian Degeneration.—This argument implies that the old neurilemmal tubes remain patent and that all the new nerve-fibres traverse these tubes. This conception is not borne out in our studies. New axons certainly grow along a graft in lines parallel to those of the old fibres, so that when growth is complete the graft is occupied by fibres running vertically and orderly from one end of the graft to the other (*see Figs. 252-255*). What provides the directing scaffolding is not certainly known. Weiss considers it to be the pattern of the colloidal ground substance of the nerve, and it is therefore of significance to note that new fibres may lie between the sheaths of the old neurilemmal tubes as well as inside them (*Fig. 258*). These fibres at least cannot be obstructed by the incompletely absorbed lipoidal products of Wallerian degeneration within the tubes; and, although we have not made special preparations to study this point, it does not appear reasonable that a fibre that can grow across a connective-tissue scar barrier at the suture lines, will find any obstruction in an intermittent lipoidal collection in the graft.

It is of significance also that Speidel^{17, 18} has shown that the myelin and neurilemma sheaths of a new nerve-fibre develop from cells in the growth-bud of the fibre itself, both sheaths thus being formed anew in regeneration.

Blocking of the Ends of the Fresh Graft by Fibrous-Tissue Reaction.—If any reaction took place it would be apparent in the sections of the suture lines between graft and nerve. It is obvious, however, in our preparations that there is no constant increase in the amount of fibrous tissue at the suture lines of the fresh graft compared with the degenerated graft (*Figs. 259-262*). Variations in the amount of scar between one experiment and another exist, but the differences appear to depend on variations in accuracy of approximation of the ends of the graft and nerve, and where accurate approximation was obtained the amount of scar at the suture line was much the same in the two types of experiment.

We have been impressed in our experiments by the influence that scar formation at the suture lines has on the passage of nerve-fibres into the distal segment of nerve. We said above that at operation we placed great importance on the technique and accuracy of suturing, aiming to pass the sutures—one on either side—through the sheath only of the nerve and graft and to approximate the cut ends of the graft to the nerve snugly and smoothly. Histological sections subsequently prepared show that we have not always been completely successful in this, and that the effect of even a small inaccuracy in approximation, or of invasion of the nerve or graft by the suture, is the production of scar tissue at the suture line, which varies in amount according to the degree of inaccuracy or invasion.

Fig. 263 represents the proximal suture line between the nerve and a 15 days' degenerated graft six days after operation, before scar tissue has begun to form. The sutures are marginally placed, and the graft is approximated to the nerve accurately and without distortion. This we look upon as the ideal operative result, and is what we aimed at—though not always successfully. Where satisfactory approximation was obtained, the amount of scar occurring 60 to 70 days later was small, and comparatively little obstruction to the downgrowth of nerve-fibres was

produced (*see Figs. 259-262*), so that in the nerve distal to the graft large numbers of axons were present (*Figs. 256, 257*), showing that the grafts had functioned efficiently.

Where, however, satisfactory approximation had not been obtained, a large scar resulted 60 to 70 days after operation. *Fig. 264*, prepared from the lower suture line of a 14-day degenerated-nerve-graft experiment 60 days after operation (*Cat 9*), shows such a result. In this experiment, the graft at operation was taken from a lower level in the left thigh than usual, and was found to be divided inside the sheath, at its lower end, into two branches. It was very difficult to approximate these two divisions accurately and smoothly to the end of the nerve, and although at the end of the operation the suturing did not appear too bad, the result 60 days



FIG. 263.—Longitudinal section of proximal suture line of degenerated auto-graft experiment. 6 days after operation. Note accurate approximation with absence of distortion. A, Proximal nerve entering anastomosis; S, Suture, marginally placed. ($\times 40$)

later was illuminating. A very extensive dense scar had formed in which large numbers of axons had become obstructed and lost, and consequently in the nerve below the graft very few fibres were present (*Fig. 265*)—in marked contrast to the condition of the nerve below the graft where scar formation at the suture lines had been minimal (*see Figs. 256, 257*).

This illustrated well the effect of scar at the suture lines on the passage of nerve-axons, and we have found in all instances where the anastomosis at operation was not perfectly satisfactory, and accurate end-to-end approximation was not obtained, that a fibrous scar formed at the suture line, in which large numbers of the growing axons became lost. Davis and Cleveland² in a recent communication have been similarly impressed by the obstructive effects of scar formation, particularly at the

distal suture line, and in a series of experiments in dogs have practised excision of the distal anastomosis and re-suture at a time when the new axons were calculated to be at the lower end of the graft: the axons, therefore, had an equal start with the connective-tissue growth in the anastomosis and could grow through into the nerve before a dense scar had formed. This method resulted in a great increase in the number of fibres reaching the distal segment of nerve.

The value of the procedure must depend on the length of graft employed. In our experiments with grafts 3 cm. in length, the amount of scar at the distal anastomosis where accurate approximation had been obtained was sufficiently small to permit the passage of the growing fibres. With longer grafts, 7 cm. in length, as used by Davis and Cleveland, the distal scar has longer time to thicken, contract, and form a relatively impassable barrier before the nerve-fibres reach the anastomosis, so that the procedure recommended by these authors would clearly be of value.



FIG. 264.—Longitudinal section of distal suture line of degenerated auto-graft experiment, 60 days after operation (*Cat 9*). Note bifurcation of nerve entering anastomosis, and extensive dense scar that has formed. A, Proximal nerve entering the anastomosis; S, Suture, marginally placed. ($\times 16$.)



FIG. 265.—Longitudinal section of nerve 6 cm. distal to anastomosis shown in Fig. 264. Full width of nerve. Note very few fibres present, and compare with Figs. 256 and 257. ($\times 135$.)

The poor clinical results recorded after the war may, we suggest, be explained on the basis of scar-tissue formation. In most of those cases the nerve gap was bridged by cable grafts—multiple strands of cutaneous nerve, often arranged around a catgut strand running between the two ends of the nerve and enclosed in a connective-tissue sheath. Our experiences suggest that under such conditions fibrosis would be so extensive as to prevent any nerve-fibres reaching the distal end of the nerve. A certain amount of scar formation at the anastomosis is inevitable, but must be kept to a minimum if a functional result is to be obtained, and we suggest that in order to ensure a satisfactory result two things are essential: firstly, meticulous care and accuracy of technique in suturing; and secondly, employment of a graft of the same calibre as the nerve, so that accurate approximation of the cut end of the graft and nerve can be obtained.

In autogenous nerve-grafting, therefore, we do not confirm Duel and Ballance's results, and find that with equal accuracy of approximation a fresh graft functions as efficiently as a degenerated one, and that the deciding factor in obtaining a successful result in nerve-grafting is the amount of scar at the anastomosis. In this last factor may lie the explanation of Duel and Ballance's results. When a nerve undergoes Wallerian degeneration in situ for ten to fourteen days, it becomes firmer and stiffer, and if a graft is taken from such a nerve its cut ends tend to remain circular and patent. A piece of fresh nerve, on the other hand, is soft and friable, and the cut ends, being of the consistency of butter, tend to become crushed and collapsed. Satisfactory approximation of graft and nerve can therefore be obtained more readily with a degenerated graft than with a fresh one, and this advantage would no doubt be particularly valuable in grafting the facial nerve in its bony groove, where the ends of the graft and nerve are simply laid against each other and coaptation by sutures is impossible.

We know from our results that the more accurate the approximation the less scar tissue is formed, and that with less scar the growth of nerve axons into the distal end of the nerve is facilitated, and we suggest this as a possible explanation of Duel and Ballance's findings.

b. HOMEIO-GRAFT.—If it is accepted that cable grafts are unsuitable for nerve-grafting and that a graft of equal calibre to that of the recipient nerve is necessary, then in the case of the limb nerves grafting becomes impracticable unless it is shown that a nerve taken from another individual can be used as a graft.

Homeio-graft experiments were carried out in seven cats, in each of which a fresh graft was placed in the left external popliteal nerve and a degenerated graft in the right. The results are shown in *Table III*.

Physiological Findings.—The earliest date at which return of motor function was found was 78 days after operation (*Cat 15*), when motor activity was present on both sides. In this experiment, however, the grafts were a little shorter than usual—2 cm. instead of 3 cm.—so that it is likely that return of function would have taken rather longer if the graft had been the usual length.

Table III.—RESULTS OF HOMEIO-GRAFT EXPERIMENTS

NO OF CAT	NUMBER OF DAYS PRELIMINARY DEGENERATION OF GRAFT ON RIGHT SIDE	NUMBER OF DAYS BETWEEN OPERATION AND SUBSEQUENT EXPOSURE AND STIMULATION OF NERVES	MOVEMENTS OF EXTENSOR TENDONS AS RESULT OF STIMULATION OF NERVE	
			Right Side (degenerated graft)	Left Side (fresh graft)
11	15	Cat died on 6th day after operation	—	—
12	10	44	—	—
13	24	58	—	—
14	14	59	—	—
15	17	78	—	—
16	14	Stimulated aseptically at 76 days	—	—
		Stimulated again at 91 days	—	—
17	14	96	—	—

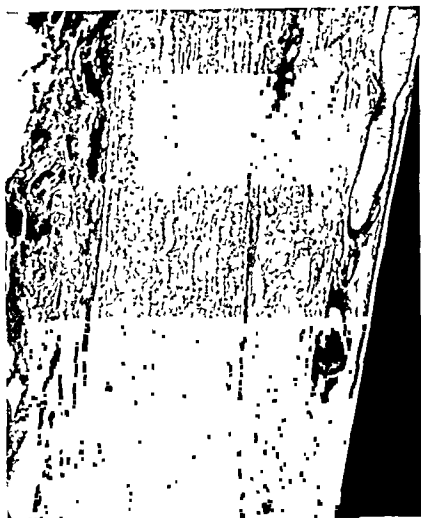


FIG. 266.—Longitudinal section of fresh homeo-graft, 96 days after operation (*Cat 17*). Full width of graft. Note: (1) Great number of fibres within graft; (2) Greatly thickened sheath full of distended blood-vessels. Compare with *Fig. 267*. ($\times 28$.)



FIG. 267.—Longitudinal section of fresh auto-graft, 88 days after operation (*Cat 5*). Full width of graft. Note: (1) Great number of fibres within graft; (2) Thin sheath with few blood-vessels. Compare with *Fig. 266*. ($\times 28$.)



FIG. 268.—Longitudinal section of nerve 6 cm. distal to homeo-graft shown in *Fig. 266*. Full width of nerve. Note presence of nerve-fibres, and compare with *Fig. 269*. ($\times 135$.)

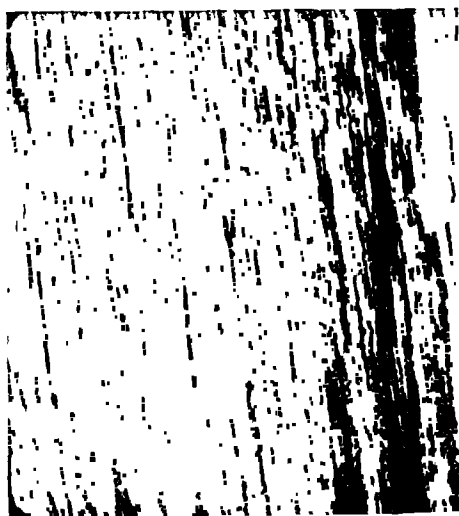


FIG. 269.—Longitudinal section of nerve 6 cm. distal to auto-graft shown in *Fig. 267*. Full width of nerve. Note presence of nerve-fibres and compare with *Fig. 268*. ($\times 135$.)

At 91 days (*Cat 16*) there was return of motor activity on the side of the fresh graft, but not on the side of the degenerated one; this cat was extremely large, and the external popliteal nerves correspondingly long, so that the distance from the upper anastomosis to the motor branch was 13.0 cm., and not the customary 10 cm., on each side, which in part explains the long interval before return of function. It is interesting to note that return of function first occurred on the side of the fresh graft. Unfortunately the nerve-axons did not take the silver stain, and it is therefore impossible to determine from the histological preparations the details and extent of nerve-fibre growth.

At 96 days (*Cat 17*) both sides showed return of motor function, so that it is evident that a homeo-graft will serve to bridge a 3-cm. nerve gap, although it appears that the nerve-fibres take longer to grow through the graft than they do through an autogenous one.



FIG. 270.—Longitudinal section of fresh autograft, 58 days after operation (*Cat 1*), counter-stained (saffronin) to show cellular constituents. Note leucocytes arranged chiefly along the strands of apparently structureless material lying between the new nerve-fibres. ($\times 135$.)



FIG. 271.—Longitudinal section of fresh autograft, 88 days after operation (*Cat 5*), counter-stained (saffronin) to show cellular constituents. Compare with Fig. 270, and note: (1) Absence of leucocytes. (2) Absence of structureless strands. (3) Graft composed of new fibres plus their cellular sheaths ($\times 135$.)

Histological Findings.—The histological findings confirm this observation. At the 60-day period nerve-fibres have passed the graft, but the fibres are of a younger type than in the corresponding auto-graft, and instead of the total distance of new axon growth being about 16 cm. it is only about 11 cm. on both fresh and degenerated sides. At 96 days, however, large numbers of fibres have passed the graft and are present in considerable numbers in the most distal segments examined (ankle level): there is no obvious difference between the extent of regeneration in this experiment and that in the 88-day fresh auto-graft (*Figs.* 266–269), so that once the original delay is overcome the final result is much the same.

The delay is due to the reaction that the homeo-graft evokes, a reaction in marked contrast to the moderate changes involving the auto-graft.

At 59 days an *auto-graft* is invaded by capillaries and leucocytes. The capillaries have grown in from the margin and branch freely within the graft; the leucocytes are full of granular material and are arranged along the lines of the nerve-fibres, and congregate especially in relation to the unabsorbed structureless strands lying between the new axons (*Fig. 270*).

Except at the suture lines, where scar tissue tends to spread intraneurally a short way into the graft and nerve, there is no invasion of the graft by connective tissue from the sheath or along the capillaries growing in from the margins.

At 73 days very few of the structureless strands within the graft remain, and the leucocytic invasion is less marked. At 88 days there is very little of the original



FIG 272—Longitudinal section of fresh homeo-graft, 59 days after operation (*Cat 14*), counter-stained (saffronin) to show cellular constituents. Note. (1) Invasion of graft by connective-tissue strands from the sheath, (2) Accumulation of large numbers of leucocytes within the graft. C, Connective-tissue strand ($\times 135$)



FIG 273—Longitudinal section of another part of the graft shown in *Fig. 272*. Note accumulation of leucocytes. ($\times 135$)

structure of the graft to be seen, leucocytes have disappeared almost completely, and the graft appears to be composed almost entirely of new fibres, each lying within its new cellular sheath (*Fig. 271*).

The appearances suggest that the original internal structure of the graft, whilst temporarily providing a scaffolding for the new nerve-fibres, is ultimately absorbed by leucocytic activity.

The *homeo-graft*, on the other hand, evokes a severe local reaction, as would be expected to result from the introduction of a foreign tissue into the animal body. Loeb has shown the stages of the reaction of host tissues to homeo- and hetero-grafts (other than nerve-grafts), which results in their destruction and absorption. These observations have been confirmed by many

workers, so that it is at first sight surprising that the introduction of a homeo-nerve-graft resulted in restoration of continuity and function of the nerve.

At the exposure of the nerve 40 or more days after operation, the homeo-graft was always much more adherent to the surrounding tissues, particularly at the suture lines, but also along its length, than in the case of the auto-graft. Sections prepared 60 days after operation show very large numbers of leucocytes within the graft, and in places growing into the graft from the sheath strands of connective tissue sometimes surrounding a small central blood-vessel. The leucocytes tend to cluster around these connective-tissue strands, and are large, swollen, and packed with granular material (*Figs. 272, 273*). This reaction is much the same in both fresh and degenerated homeo-grafts.



FIG. 274.—Longitudinal section of fresh homeo-graft 96 days after operation (*Cat 17*), counter-stained (safronin) to show cellular constituents. Note: (1) Reduction in number of leucocytes compared with 59-day homogenous graft (*Fig. 272*); (2) Absence of invading connective-tissue strands, (3) Graft composed of new fibres plus their cellular sheaths. Compare with *Fig. 271*. (135.,

In addition, aggregations of large numbers of cells occur particularly at the suture lines and under the sheath of the graft. Each aggregation consists of a hundred or more round structureless cells; the appearances are those of collections of dead leucocytes, but their localized nature, almost suggesting multiple minute abscesses, is hard to explain.

At 96 days this marked foreign-body reaction is considerably modified (*Fig. 274*). Large numbers of active granular leucocytes are still within the graft, but the invading connective-tissue strands have gone; there is, too, little to be seen of the original structure of the graft, which appears to be composed entirely of nerve-axons each with its new cellular sheath. Apart from the large number of leucocytes still within the graft the condition closely resembles that of the auto-graft. The aggregations of cells beneath the sheath are still present, but are becoming organized.

It is thus seen that the foreign tissue of the graft is partly absorbed by leucocytic activity and partly replaced by connective-tissue invasion, but that this invasion by fixed tissue cells is in its turn absorbed, and the final structure of the graft appears to be made up of a new growth from the proximal end of the nerve, as in the case of the auto-graft.

Although the internal structure of the graft is absorbed in this way, it must in its early stages provide the necessary scaffolding for the new growing fibres; it is obvious, however, that the connective-tissue invasion will hold up their growth, and this must in part be responsible for the delay in passage of fibres through the homeo-graft compared with the auto-graft. But the delay must also be due in part to reaction at the suture lines, where a marked connective-tissue reaction takes place which is not absorbed and forms an appreciable scar at the anastomosis. This scarring is in considerable excess of that occurring in the auto-graft experiments, where good approximation was obtained. In our experiments with grafts 3 cm. long, fibres are able to pass into the distal end of the nerve in as great numbers as through the auto-graft, although their passage is delayed. With a graft three times as long, however, the corresponding increase in length of connective-tissue-invaded graft through which the fibres must grow, plus the scarring at the suture lines, might prove a considerable barrier to nerve-axon growth. We must conclude, therefore, that though a homeo-graft will bridge successfully a short nerve gap, the fate of the growth of nerve-fibres through a long homeo-graft still requires experimental investigation.

SUMMARY

1. Nerve-suture and nerve-grafting experiments have been performed in the cat.
2. It is shown that a nerve gap 3 cm. in length can be bridged by a nerve-graft so that the anatomical and physiological continuity of the nerve is restored.
3. The advantages claimed for a degenerated nerve-graft over a fresh graft are not confirmed, and it is found that the results after fresh- and degenerated-nerve-grafting are indistinguishable.
4. The reduction of scar tissue at the suture lines is shown to be of the greatest importance in obtaining a successful result.
5. In order to reduce scar tissue to a minimum, two factors are considered to be essential: firstly, accuracy of approximation of nerve and graft; and secondly (a corollary of the first), a graft of equal calibre to the recipient nerve.
6. Nerve gaps 3 cm. in length have been satisfactorily bridged by homeo-nerve-grafts, but it is not possible to say whether longer grafts would function as efficiently.

The authors wish to acknowledge their indebtedness to the late Sir Charles Ballance, with whom this investigation was begun. Although Sir Charles's illness soon caused him to stop active work, the present authors owe a great deal to the stimulus of his enthusiastic and critical mind. They wish also to express their thanks to Mr. S. P. Steward, Mr. F. Watson, and the Technical Staff of the Royal College of Surgeons Laboratories for their willing and skilful help throughout this work.

REFERENCES

- ¹ STOOKEY, *Surgical and Mechanical Treatment of Peripheral Nerves*, 1922. Philadelphia : Saunders.
- ² DAVIS and CLEVELAND, *Ann. of Surg.*, 1934, Feb., 271.
- ³ NAGEOTTE, *Revue neurol.*, 1920, xxvii, 1131.
- ⁴ SENCERT, *Presse méd.*, 1918, xxvi, 656.
- ⁵ DUSTIN, *Comptes rend. Soc. de Biol. de Paris*, 1919, lxxxii, 614.
- ⁶ STOPFORD, *Lancet*, 1920, ii, 1296.
- ⁷ PLATT, *Brit. Jour. Surg.*, 1919-20, vii, 384.
- ⁸ PLATT and BRISTOW, *Ibid.*, 1923-24, xi, 535.
- ⁹ DUEL and BALLANCE, *Arch. of Otolaryngol.*, 1932, xv, 1.
- ¹⁰ DUEL and BALLANCE, *Ibid.*, xvi, 767.
- ¹¹ TELLO, *Trab. del Lab. de Investig. Biol.*, 1911, ix, 123.
- ¹² CAJAL, *Degeneration and Regeneration of the Nervous System*, 1928, i, 361.
- ¹³ BALLANCE, *Jour. Laryngol. and Otol.*, 1934, xlix, 709.
- ¹⁴ DUEL, *Surg. Gynecol. and Obst.*, 1933, lvi, 382.
- ¹⁵ DUEL, *Brit. Med. Jour.*, 1934, ii, 1027.
- ¹⁶ WEISS, *Jour. of Exper. Zool.*, lxviii, 293.
- ¹⁷ SPEIDEL, *Ibid.*, lxi, 279.
- ¹⁸ SPEIDEL, *Amer. Jour. Anat.*, 1933, lii, 1.

AN EXPERIMENTAL STUDY OF THE RETURN OF FUNCTION AFTER TENDON SECTION

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INTRODUCTION

DURING recent years the natural processes by which cut tendons are repaired have interested a large number of Continental workers, and a considerable literature has grown up around this subject. Very little of this work has appeared in the English language, and it would therefore seem that the problem has not been very closely studied by either British or American workers. Mason and Shearon (1932) published a paper on the process of tendon repair in which they included an extensive survey of the Continental work. An excellent account of the early work was given by Adams (1860) in his book on the reparative processes in human tendons, which laid the solid foundations of our knowledge of tendon repair. It is therefore not considered necessary in this communication to give a formal review of the literature, which would occupy more space than would be justified in a paper of this nature. Reference will only be made to those papers which have a direct bearing upon the aspects of the problem which have been investigated in the present inquiry.

The present research arose out of an investigation which the author had conducted by means of slow motion cinematography into the alterations which occur in the gait after the tendons of various muscles have been cut. In that investigation, the author used rabbits, guinea-pigs, and cats, and the tendons divided were those of the distal region of the hind limb. Two guinea-pigs, in which all the tendons on the posterior aspect of both the hind limbs immediately above the ankle had been severed, were kept for several months, when it was discovered that their gait had practically returned to the normal. The animals were killed and post-mortem examinations were made. Macroscopically, it was found that the divided tendons had been re-formed, and histological examination revealed that the newly formed tissue closely resembled normal tendon tissue in its structure. This discovery appeared to justify a special investigation into this aspect of tendon repair, and a series of experiments was performed upon three cats (Nos. 3, 6, and 7). These experiments form the basis of the present communication. The cat was selected as the experimental animal because, being digitigrade, section of the muscles of the region of its calf causes much greater alteration in its gait than a similar operation on the plantigrade guinea-pig.

TECHNIQUE

The animal was anæsthetized and an incision was made, with the usual aseptic precautions, along the inner border of the superficial tendons of the foot immediately above the heel. The sheath of these tendons was cleaned, opened, and the

structures within it exposed. In the cat these structures are two in number—the tendo Achillis, formed by the fusion of the tendons of the gastrocnemius and the soleus, and the tendon of the large plantaris muscle, which is a separate entity. After they had been exposed, about half an inch of each of the tendons was removed and the muscle bellies were allowed to retract. It was difficult to estimate the exact length of the gap between the ends of the tendons after the retraction, but it was about $\frac{3}{4}$ to 1 in. in length. The deep tendons were next exposed and divided. There was practically no bleeding during the operation, and after the deep tendons had been divided the skin incision was closed with horsehair sutures. The operation was then repeated on the other leg. The animals received no further treatment, and were merely confined in a large room in which they could move about quite freely. There was no sign of sepsis during the recovery period and the wounds healed by first intention.

CINEMATOGRAPH ANALYSIS

Before the operations were performed, a 16-mm. cinematograph film was made of the normal gait of the animals. A week was allowed to elapse after operation to allow the animals to recover from post-operative stiffness, and a further film was made. At the end of the second week, the gait of the animals resembled so closely that at the preceding week that it appeared to be unnecessary to make further pictures at this stage and cinematography was therefore postponed till the end of the third week. After this, the films were made at weekly intervals—namely, at the fourth, fifth, and sixth week. At the end of this period the experiments were concluded, by sacrificing the animals. To facilitate the analysis of the variations in the gait of the animals at the different phases of the experiments, the films were taken at the rate of 64 frames per second. Therefore, when they were projected on the screen, the animals appeared to be walking at a quarter the normal rate.

In presenting accounts of research work involving cinematography, authors labour under the handicap that the readers are not able to see the actual film and may therefore have some difficulty in following the description. To overcome this obstacle to a certain extent, I have made a series of outline drawings of the hind-quarters of one of the animals by projecting the film on to a roll of paper and tracing the position of the limbs in every third frame throughout a complete cycle of movement (*Fig. 275*).

It is customary to divide the locomotory cycle of a limb into two parts—(1) the support stage, when the limb is in contact with the ground and is supporting the weight of the animal, and (2) the suspense stage, when the limb is off the ground.

Normal Gait (*Fig. 275a*).—The normal gait of the cat is definitely digitigrade, and at no part of the support stage does the posterior portion of the foot come in contact with the ground. At the start of this stage, only the toe region of the foot reaches the ground and the heel is well raised from it. As the weight of the animal is brought on to the supporting limb, the heel is gradually lowered, but is always well above the horizontal. In the latter part of the support stage, the heel gradually rises to and beyond the perpendicular. At the start of the suspense period, the foot at first inclines still further beyond the perpendicular, but very rapidly returns to that position. As it is carried forward in the latter part of suspense, the fore-part of the foot becomes raised, and the whole foot comes nearer to the horizontal than at any other point in the locomotory cycle.

Gait One Week after Operation (*Fig. 275b*).—The animal is now completely plantigrade during the greater part of the support period. At the start of this stage, the fore-part of the foot may first of all make contact with the ground, but if this happens it is immediately followed by the remainder of the foot. On the other hand, the whole of the plantar surface of the foot may come to the ground at one time. During the greater part of the support period, the whole of the foot remains in contact with the ground, and only towards the end of this stage is the heel raised from the ground. This raising of the heel appears to be distinctly passive, due to the animal being pulled forward by its fore-limbs with little active

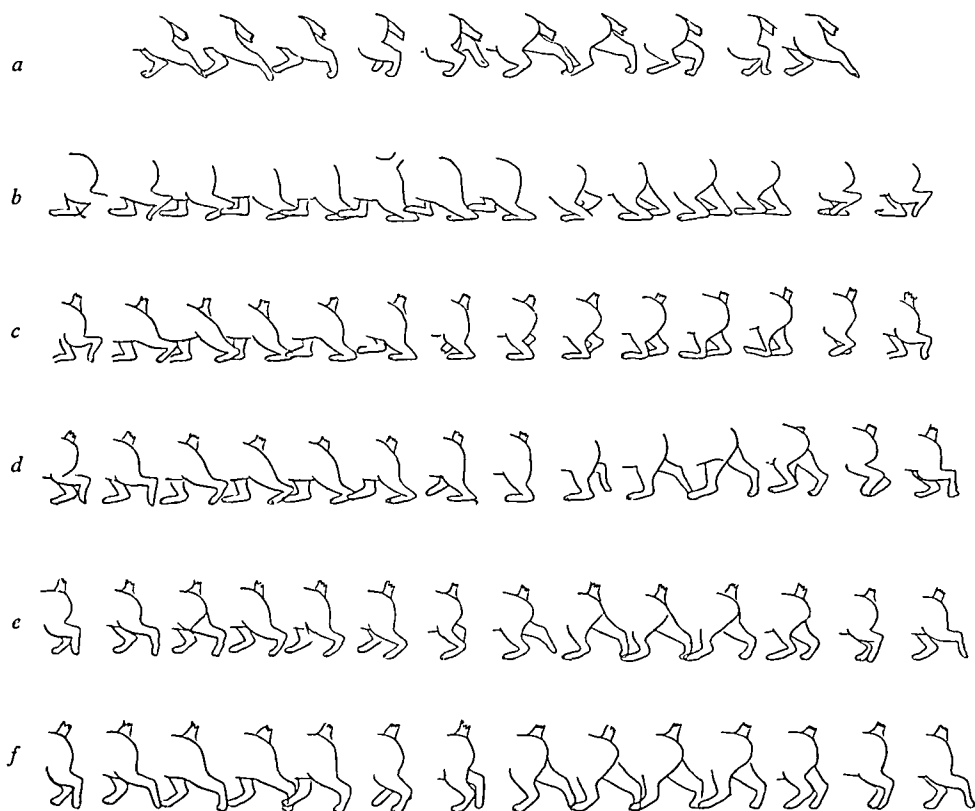


FIG. 275.—Tracings of gaits of cat from cinematographic films. *a*, Before operation; *b*, One week after operation; *c*, Three weeks after operation; *d*, Four weeks after operation; *e*, Five weeks after operation; *f*, Six weeks after operation. The tracings start from the right of the figure.

assistance from the muscles of the leg. The foot is rolled forward and does not reach the perpendicular position. The suspense period begins by the foot being lifted from the ground by the action of the thigh muscles. It is then carried forward, but to a much less extent and less efficiently than in the normal animal. In the suspense period the fore-part of the foot is held relatively much higher than in the normal animal, so that the toes are at the same height as the heel or may even be raised above it. The action of the whole of the hind-quarters region is stiff and inefficient.

Gait Three Weeks after Operation (Fig. 275c).—Although the animal is still completely plantigrade during the greater part of the support period, the hind-limbs move more freely and their muscles are taking a much more active part in the movements than they did a week after operation. The limb below the knee is now more perpendicular during the earlier stage of support, indicating that it is resisting the weight of the animal and is now not passively yielding. During suspense, the hind-limb appears actively to assist the carrying forward of the foot, and this movement is now more extensive.

Gait Four Weeks after Operation (Fig. 275d).—There is a very marked improvement in the gait and the movements of the hind-limbs are much freer. At the start of support the fore-part of the foot of the animal makes contact with the ground, but the calcaneal region sags very rapidly and the fully plantigrade position is reached. During the latter part of the support period, the heel is raised from the ground by muscular action and is not passively rolled upwards as before. At the end of support and the start of suspension, the foot approaches nearer to the perpendicular, and when it is carried forward the heel is always higher than the toes.

Gait Five Weeks after Operation (Fig. 275e).—The gait has now almost resumed its normal form. Close observation, however, shows that at the start of support more of the anterior portion of the foot is on the ground than in the normal animal. Also, as the weight comes on to the foot, the heel approaches more closely to the ground than before operation.

Gait Six Weeks after Operation (Fig. 275f).—The gait appears to be exactly similar to that of the normal animal.

During the period of six weeks that the animals were kept after operation, the hind-quarters were examined at frequent intervals. At first there was no resistance at the site of operation and the posterior surface of the bone could be palpated easily through the skin. Later, there occurred a steady thickening at the site of operation and an increasing resistance to the fingers. Towards the end of the period, a definite rounded strand similar to a tendon could be felt. The skin was freely movable over this structure.

The critical stage of these experiments was during the fourth week after operation, and it is obvious that at this time sufficient regeneration had occurred to allow the divided muscles to recommence functioning. There is an interesting parallel to this result in the work of Gaza, Gerlach, and Gissel (1932), who studied the metabolic changes in the regenerating tissue at different stages of tendon regeneration. They found that after tendon section, the metabolic activity of the tissue uniting the ends of the tendons steadily increased up to 18 days. After this it gradually sank until it reached that of normal tendon. From the results of these workers and those of the present research, it would appear that some marked change takes place about the third week in the tissue filling the gap between the tendons, and it is suggested that this may be the commencing transformation of some tissue, resembling granulation tissue, into tendon. This is supported by the work of Bloch and Bonnet (1929), who found histologically that towards the end of the third week the tissue filling the gap began to resemble tendon.

POST-MORTEM RESULTS

After six weeks the cats were killed and a post-mortem examination was made. For comparison, a similar examination was done on a normal cat (Fig. 276).

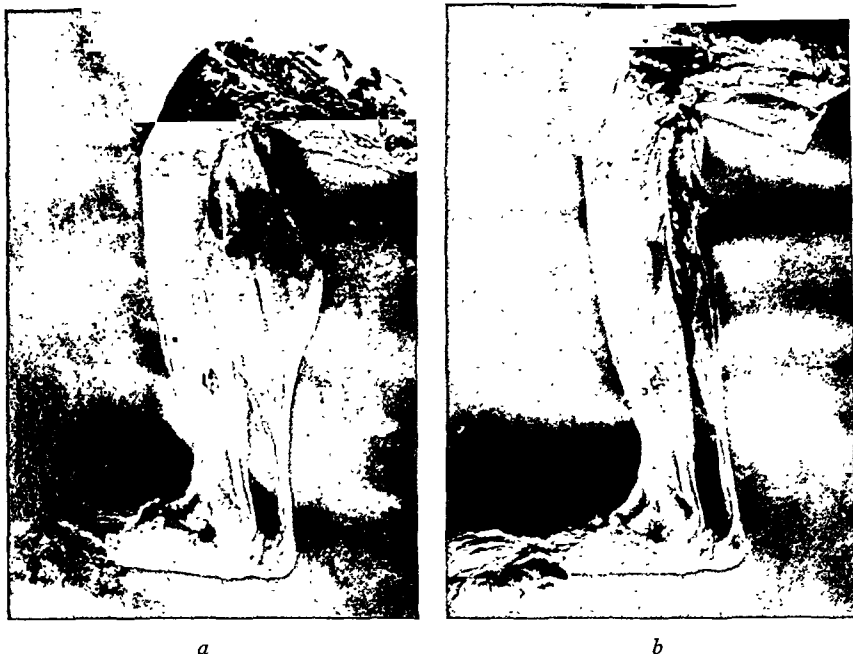


FIG. 276.—Dissections of the hind limb of normal cat. *a*, The skin and fascia have been removed and the superficial muscles and tendons cleaned; *b*, As (*a*), but the gastrocnemius muscle and the tendo Achillis have been removed to expose the plantaris muscle and tendon.



FIG. 277.—Experimental Cat 3, six weeks after operation. The dissections are the same as those done on the normal cat shown in Fig. 276, *a* and *b*, except that only the belly of the gastrocnemius could be removed as there is no separate tendo Achillis. Gastrocnemius, plantaris, and soleus have now a common tendon.

Cat 3.—There is a complete continuity of structure between the cut ends of the tendon of the plantaris. The two heads of the gastrocnemius are markedly shortened and relaxed, and their cut ends are attached to the belly of the plantaris. They cannot have been of much assistance in raising the heel. The newly formed tendon is directly continuous with the plantaris and is proportionately longer than the original tendon. The soleus is also attached to the new tendon. The deep flexors have reunited (*Fig. 277.*)

Cat 7.—This closely resembles the condition seen in *Cat 3*. The newly formed tendon is, however, less firm and more diffuse. The gait of this animal did not recover so completely as *Cats 3* and *6* and this was probably due to the condition of the newly formed tendon. This less complete recovery can probably be explained by the general condition of the animal, which was rather emaciated and suffered from diarrhœa during the whole period of experiments.



FIG. 278.—Experimental Cat 6. Similar to *Fig. 277, a* and *b*.

Cat 6.—This showed a much better anatomical result than was seen in the other animals. The new tendon could be partially divided into two parts, one formed from the gastrocnemius and a portion of the plantaris, and the other from the remainder of the plantaris and soleus. Although less marked than in the previous animals, the newly formed tendon was relatively longer than the normal and the muscle bellies had shortened. The deep flexors had reunited. (*Fig. 278.*)

In all the animals the tendons had definite sheaths within which they were freely movable.

The results of these experiments do not agree with the views of Wehner (1923). That investigator states that the ability of the newly formed tendon to function depends upon the position taken up by the joint during recovery. If this is outside the normal pose of the animal, the regenerated tendon will be too long.

On account of this the contractile power of the muscle will be diminished and there will be functional insufficiency of the tendon. In the present investigation, the pose of the limb after the operation was undoubtedly quite different from the normal of the cat. This caused a lengthening of the newly formed tendon and a shortening of the muscle, which was marked in two of the animals. This, however, did not lead to any disability, and it must be presumed that the muscle was able to function quite satisfactorily in its new position.

HISTOLOGICAL RESULTS

The tendons were removed, embedded in celloidin, and histological sections were prepared stained with hæmalum and eosin (*Fig. 279*). At the same time

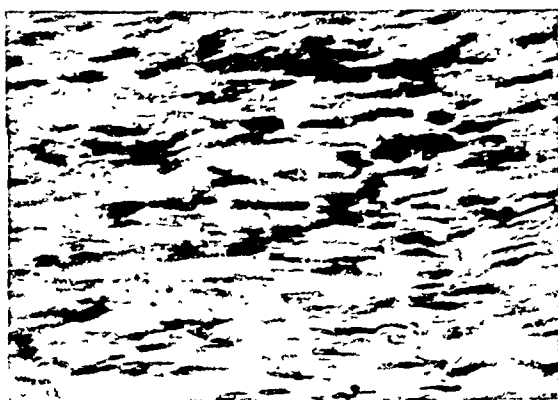


FIG. 279.—Microscopical section of regenerated tendon of cat.

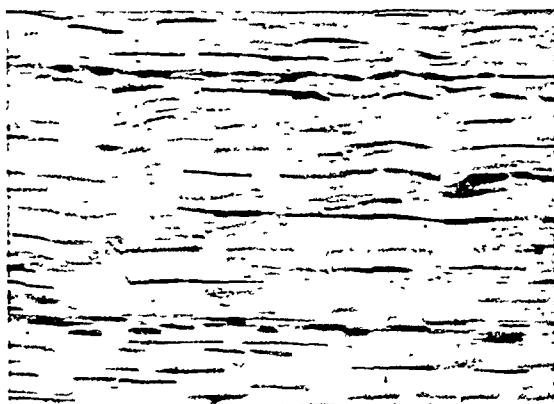


FIG. 280.—Microscopical section of normal tendon.

tendons were taken from the same region of a normal cat and similar histological preparations were made of these for purposes of comparison (*Fig. 280*).

The newly formed tissue is very definitely tendinous in appearance microscopically, but shows several features which distinguish it from the normal tendon. The fibres, though tending to be parallel, do not all run in the same direction, and they also differ from the normal as their course is rather waved instead of being in straight lines. This waviness is also seen in the direction of the cells, which are also much less compressed from side to side than in the normal tendon. Although they are arranged in rows, these are not so clearly defined as in the normal condition, neither are the rows so markedly separated from each other by the fibrous bundles, which are much narrower in the newly formed tissue.

These histological findings agree with the results obtained by the great majority of workers in this field. It is true that certain investigators, such as Narvi (1931), believe that the gap is filled with scar tissue which in no way resembles tendon, but such negative conclusions cannot be given much consideration when weighed against the large volume of positive evidence that the newly formed tissue is either tendon or a tissue which structurally resembles tendon.

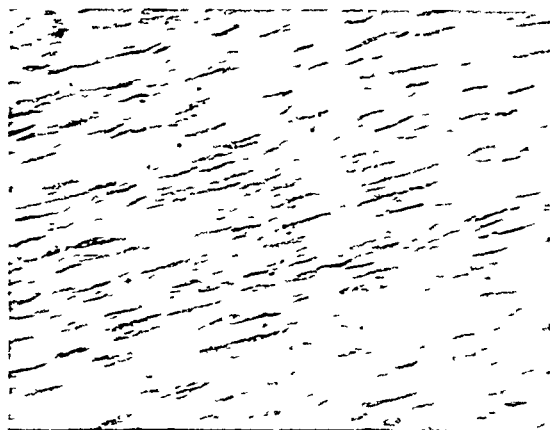


FIG 281.—Regenerated tendon of guinea-pig, four months after operation.

The present experiments, on account of the shortness of their duration, do not materially assist in elucidating this problem as to whether the newly formed tissue is true tendon or merely a tissue which has come to resemble tendon on account of the strains and stresses to which it is subjected. The histological preparations undoubtedly resemble tendon, but, as has already been stated, they have definite characters by which they can be differentiated from ordinary tendon. It is possible that if the animals had been preserved for a longer period the distinguishing features might have disappeared. However, the author can throw some further light on this problem. A guinea-pig which underwent a similar operation to that performed upon the cats, was kept alive for four months after the operation. It will be observed that regenerated tissue in this animal (*Fig. 281*) resembles normal tendon much more closely than the same tissue in the cats which had only been kept alive for a period of six weeks. It would therefore seem probable that the tissue which reunites the ends of the tendons in the course of time approximates more and more closely to the structure of normal tendon.

SUMMARY AND CONCLUSIONS

In three cats about half an inch of the tendons of the superficial plantar flexor muscles was removed and the tendons of the deep flexors were divided. The sheaths were preserved. The animals were allowed to move about freely without further treatment. Six weeks after the operation the animals' gaits had returned to normal. Histological preparations were made from the new tissue, and this was found to have many of the characteristics of normal tendon.

I have to thank Professors Raper and Stopford for facilities to carry out this work in the Departments of Physiology and Anatomy of Manchester University.

The cost of this investigation has been met by a grant from the Manchester University Medical Research Fund.

REFERENCES

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- ADAMS, W., *The Reparative Process in Human Tendons*, 1860. London : John Churchill.
BLOCH and BONNET, *Congrès française de Chir.*, 1929, 547.
GAZA, GERLACH, and GISSEL, *Arch. f. klin. Chir.*, 1932, clxx, 19.
MASON and SHEARON, *Arch. of Surg.*, 1932, xxv, 615.
NARVI, E. J., *Acta chir. Scand.*, 1931, xviii, 267.
WEHNER, E., *Deut. Zeits. f. Chir.*, 1923, clxxvii, 169.

*SHORT NOTES OF RARE OR OBSCURE CASES***SQUAMOUS-CELL CARCINOMA OF THE RENAL PELVIS
IN A HORSESHOE KIDNEY**

BY W. F. NICHOLSON, MANCHESTER

SQUAMOUS-CELL carcinoma of the kidney is uncommon, but to find it associated with horseshoe kidney is sufficiently rare to justify recording this case.

The patient was a man of 51, who on admission to the Manchester Royal Infirmary gave a history of recurrent attacks of pain in the left iliac fossa, associated with hæmaturia for four months. Cystoscopy revealed blood spurting from the left ureteric orifice, and uroselectan radiography suggested the presence of a horseshoe kidney. The latter was confirmed by ascending pyelograms with sodium iodide, the radiograph showing dilatation and irregularity of the left renal pelvis



FIG. 282.—High-power view, showing growth invading kidney.

in addition. Apart from culture of *Sta. albus* from the urine, the urinary function was normal. A diagnosis of neoplasm in the left half of a horseshoe kidney was made, and in view of the repeated hæmaturia prompt exploration was advised.

Mr. Graham Bryce explored the abdomen through a left paramedian incision, the descending colon being mobilized and reflected towards the midline. The left renal pelvis was dilated, and there was hydro-ureter down to the level of the

isthmus of the horseshoe, where the ureters crossed in front of the renal substance. Nephrectomy of the left kidney was performed, the isthmus being divided at the



FIG. 283—Low-power view of renal pelvis, showing proliferation of epithelium in parts and degeneration in other parts



FIG. 284—Low-power view showing renal substance invaded by growth

lower pole of the left kidney. On naked-eye examination the left kidney was hydro-nephrotic with much fibrosis of the pelvis. No obvious growth could be identified, but the whole pelvis was greatly thickened.

Microscopical examination showed that this was a squamous-cell carcinoma arising in the pelvis and invading the kidney substance (*Figs. 282-284*). The patient made a good recovery from operation, but died of secondary deposits six months later.

Comment.—In a review of 68 cases of horseshoe kidney, Walters and Priestley¹ at the Mayo Clinic described only three tumours, none of them squamous carcinoma. Similarly Rathburn² described four neoplasms in 108 cases of horseshoe kidney, but none of them was squamous carcinoma. The microscopical sections show the lack of cell-nests which is the feature of squamous carcinoma in this region, to which Silverstone³ has recently called attention. The pelvic epithelium has proliferated in places, but mostly is degenerate and shows signs of chronic pyelitis. Nowhere does it show definite leukoplakia, as Kutzmann⁴ and Thomson-Walker⁵ have described in association with these growths.

I am indebted to Mr. Graham Bryce for encouragement and permission to publish the case, and to Dr. Loveday for the sections.

REFERENCES

- ¹ WALTMAN WALTERS and JOHN PRIESTLEY, *Jour. of Urol.*, 1932, xxviii, 271.
- ² RATHBURN, *Ibid.*, 1924, xii, 611.
- ³ MAURICE SILVERSTONE, *Brit. Jour. Surg.*, 1935, Oct., 332.
- ⁴ KUTZMANN, *Arch. of Surg.*, 1929, xix, 871.
- ⁵ THOMSON-WALKER, *Proc. Roy. Soc. Med.*, 1927, xx, 720.

B. WELCHII INFECTION COMPLICATING CONSERVATIVELY TREATED APPENDICEAL ABSCESS

By STUART GORDON, TORONTO

The following case report is considered to be of sufficient interest to justify publication.

O. B., a farmer aged 50, was admitted to the Toronto General Hospital on Oct. 25, 1935. Three days previously he had first complained of abdominal discomfort. Two days prior to admission, the discomfort shifted to the right lower quadrant of the abdomen. He lost his appetite. During the following day he became nauseated and vomited. His pain became very severe and he called his doctor at midnight. Examination at 4 a.m. revealed a spare individual who did not appear ill. The lower part of the abdominal wall did not move with inspiration and expiration. There was hyperæsthesia over the right lower quadrant. The right rectus was splinted. A tender mass about the size of a lemon was palpable in the region of the cæcum. Rectal examination revealed a tender area high and to the right. His temperature was 101.8° by mouth; his leucocyte count 20,000. The urine was normal. A provisional diagnosis of appendiceal abscess was made and conservative treatment decided upon. The patient accordingly was placed in Fowler's position, continuous intravenous saline was started, and an electric cradle was placed over the abdomen. Ice chips only were allowed by mouth; no laxative, no enema, and no sedative. A close watch was kept on the pulse-rate.

Eight hours later the abdomen was soft and the patient appeared comfortable. For the next two days his condition was quite satisfactory. On Oct. 28, following

an examination by a medical student, the patient stated he definitely and suddenly became worse. His temperature rose to 102° . The abdomen during the next few hours became distended, tender wherever examined, and the heart-sounds could be detected by auscultation in the lower half of the abdomen. It was believed that the abscess had ruptured and that the patient was now suffering from a general peritonitis. Four-hourly injections of pituitrin and eserine were ordered. These were effectual. By the evening of Oct. 29 his condition had improved considerably. The abdominal tenderness had lessened markedly. There was no distension. Heart-sounds could no longer be heard over the abdomen. A large pelvic abscess was felt on rectal examination.

The patient was seen at 7 a.m. on the morning of Oct. 30 by the house surgeon. He complained of numbness in the right thigh, but examination showed nothing

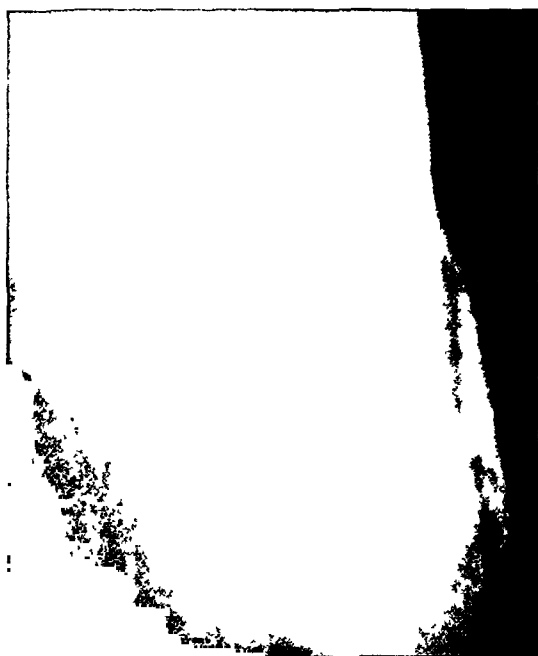


FIG. 285.—Radiograph showing extensive gaseous infiltration of the upper part of the thigh.

abnormal. At 9 a.m. the patient was again seen. False emphysema was noted in Scarpa's triangle. Gas gangrene serum was given intramuscularly. By mid-afternoon the thigh was twice its normal size, there were large dark bluish blebs along its medial aspect, and gangrenous emphysema was present from inguinal ligament to knee. Gas gangrene serum was again administered, this time intravenously, and the patient taken to the anæsthetic room. Under gas-oxygen anaesthesia four incisions were made the length of the right thigh. Foul-smelling gas hissed and bubbled out behind the scalpel. The adductor muscles were completely destroyed, being replaced by a structureless dull-red mass. Four rubber tubes were pushed for varying distances into the thigh and connected with an oxygen tank. Cultures were taken. Shortly afterwards the patient died.

At post-mortem a large appendiceal abscess was found. The appendix had perforated at its base and extruded a fæcolith. A probe was passed without any difficulty into the cæcum. The lower boundary of the abscess was formed by the peritoneum overlying the iliacus muscle. An area the size of a shilling had been eroded in the peritoneum and fascia overlying the muscle. Bubbles of gas were present in the iliacus muscle. It would appear, therefore, that the infection had spread by way of the iliacus into the thigh. There was a mild general peritonitis, and a large pelvic abscess. The gaseous infiltration in the thigh is shown in Figs. 285, 286.



FIG. 286.—Showing the infiltration of the lower part of the thigh.

The culture taken at operation revealed *B. welchii*, as did a culture taken from the right thigh at post-mortem. Cultures of the peritoneal cavity and the pelvic and appendiceal abscesses grew a streptococcus.

Summary.—A case of appendiceal abscess, treated conservatively, which resulted in a gas gangrene infection with fatal termination, is reported. It is presumed that the organism responsible for the infection spread from cæcum to abscess, to iliacus muscle, and to thigh. The rarity of the complication, together with the fact that no operative interference had taken place prior to the development of the gas bacillus infection, are considered sufficient justification for this communication.

UMBILICAL FISTULA CAUSED BY A PATENT MECKEL'S DIVERTICULUM

By MAY RATNAYEKE

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CASES of persistent Meckel's diverticulum with external opening are sufficiently rare to warrant publication.

Meckel's diverticulum is a persistent remnant of the omphalomesenteric duct. It is represented by a mere pouch in most cases; occasionally it may extend from the ileum to the umbilicus, or float free at the distal end. In this case the diverticulum was patent and produced a fistula discharging liquid yellow faecal matter and gas.

HISTORY.—A male infant 6 weeks old was admitted to the Lady Ridgeway Hospital, Colombo, with a history of a discharge of fluid and faeculent material through a red polypoid protrusion at the umbilicus since the separation of the umbilical cord on the tenth day after birth.

PHYSICAL EXAMINATION.—The infant is well nourished. The stools are normal, one or two a day. The skin around the umbilicus is excoriated. At the umbilicus there is a protrusion of mucous membrane discharging faeculent material and gas when the child strains or when the abdomen is stroked. The child is breast-fed. Its weight is 9 lb. 5½ oz. An X-ray examination after injection of 2 c.c. of lipiodol through the fistula displayed the track of the fistula and the lipiodol outlining the coils of small intestine.

FIG. 287.—Showing the incision encircling the umbilical fistula.

OPERATION.—The infant was operated on under open ether anaesthesia on Feb. 25, 1936. An incision was made around the umbilicus with its mucous protrusion (*Fig. 287*) and deepened until the peritoneum was seen encircling the diverticulum of gut. In order to deal with the diverticulum at its origin, the peritoneum was opened up about an inch

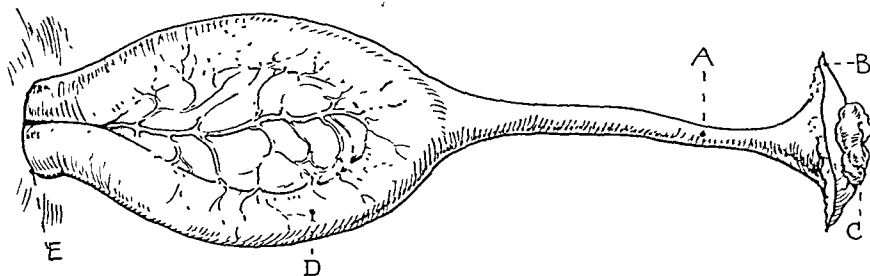


FIG. 288.—The diverticulum and the loop of gut withdrawn from the abdomen. A, Meckel's diverticulum; B, Umbilicus dissected out; C, Protrusion of mucous membrane; D, Loop of gut; E, Incision in parietes.

and the loop of intestine from which the diverticulum arose was lifted out by traction on the diverticulum (*Fig. 288*). When this was exposed it was found to be 3 in. in length, and had the appearance of a normal appendix. The base of the diverticulum was clamped, crushed, ligated, and divided, not too close to

the gut, in order to avoid narrowing of the gut lumen. The stump was not buried, but two occluding sutures partially covered its stump (*Fig. 289*). The

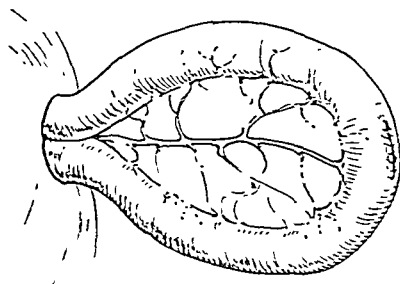


FIG. 289.—The diverticulum has been removed and the gut sutured.

intestine was returned to the abdomen. The opening in the peritoneum was closed, the rectus was approximated, and the skin edges were brought together.

The child made an uneventful recovery, and was discharged cured on the twenty-first day.

AN INACTIVE PARATHYROID TUMOUR

BY GEOFFREY KEYNES

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A LARGE number of examples of enlargement of the parathyroid glands have now been reported in surgical literature, interest in them being due not to their features as tumours in the neck, but solely to their influence on the skeleton through their internal secretion. They are indeed scarcely to be regarded as 'tumours' in the pathological sense, being rather due to enlargement of normal parathyroid bodies, without production of any abnormal tissue. Their structure is that of normal parathyroid tissue, and it is very seldom that any swelling can be detected clinically in the neck or elsewhere. Usually the presence of the parathyroid enlargement is inferred from its endocrine effects, and an exploratory operation is undertaken in the neck or mediastinum to discover its whereabouts. It may be of some interest, therefore, to describe a rare condition in which the position is reversed, an operation being undertaken to remove a large and clinically palpable parathyroid tumour which had not produced any endocrine effects whatever.

A female patient, aged 50, was referred to me recently by Dr. Hinds Howell for an opinion concerning a tumour in the neck. On the left side of the neck was a firm solid swelling, situated beneath the upper part of the sternomastoid, close to, and superficial to, the upper pole of the thyroid gland. It was not easy to feel, being mobile and deeply placed. No definite diagnosis could be made, since it was producing no symptoms; nevertheless removal seemed to be advisable on general principles.

The tumour was easily removed from the upper deep cervical region through a high collar incision, and it did not have any obvious attachment to the parotid, the thyroid, or any of the neighbouring structures.

The tumour was of irregular shape, of homogeneous consistency, with a whitish cut surface, and weighed 18 g. The microscopical section showed a solid cellular

structure resembling that of a parathyroid gland. It was, in fact, impossible to regard the tumour as being anything but a parathyroid adenoma, and this opinion was confirmed by Professor Geoffrey Hadfield, who pointed out, however, that the tumour consisted almost exclusively of the oxyphil cells usually seen in small groups in sections of normal parathyroid glands. This fact is demonstrated in the accompanying figure of a typical area made from a colour-photograph taken in Professor Hadfield's laboratory (Fig. 290).

Radiograms of the patient's skeleton were taken after the nature of the tumour had been established, but no abnormality was discovered. Estimation of her serum calcium was not considered worth while, since the level of this would in any case have fallen to normal immediately after the removal of an active parathyroid tumour.

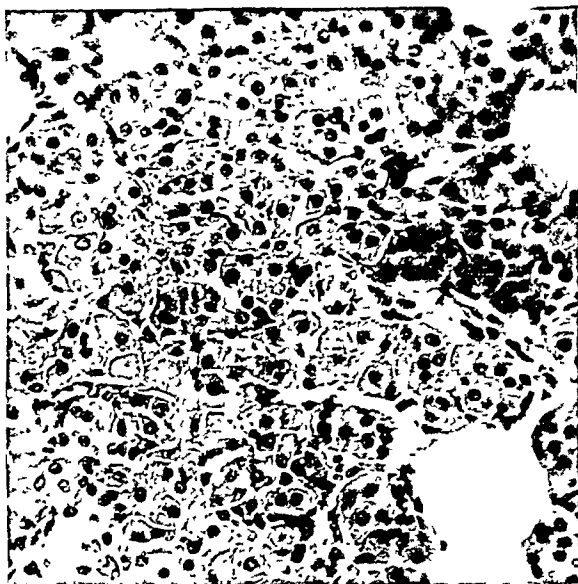


FIG. 290.—Section through tumour, showing solid cellular structure resembling parathyroid gland, but consisting almost entirely of oxyphil cells. ($\times 170$.)

Comment.—This case seems to establish the occasional occurrence of a large parathyroid tumour in the neck which is totally inactive as regards its internal secretion. Hadfield and Rogers¹ have already recorded such a tumour, weighing 52 g., in a woman of 58 whose skeleton was normal radiologically, and report that three others have been described by Hoffheinz.² Professor Hadfield³ has also made the suggestion that the lack of secretory activity in the tumour here recorded, and perhaps in others, is due to the special character of the cells composing it. He informs me that the tumour in his case mentioned above was also composed almost entirely of oxyphil cells, this feature probably indicating that they are in a senile or inactive phase. The same interpretation is to be put upon the groups of similar cells seen in the normal gland.

REFERENCES

- ¹ HADFIELD, G., and ROGERS, H., *Jour. Pathol. and Bacteriol.*, 1932, xxxv, 259.
- ² HOFFHEINZ, *Virchow's Arch. f. pathol. Anat.*, 1925, cclvi, 705.
- ³ Personal communication.

SMALL ANGIOMA OF RENAL CALIX

By H. A. COOKSON

HON. PATHOLOGIST TO SUNDERLAND ROYAL INFIRMARY

A FEW notes on a case of hæmaturia are given, because the case illustrates how in the past it would have been grouped under the heading of 'renal hæmophilia' or 'essential hæmaturia'.

H. P., a single woman, aged 36 years, a shop assistant, was admitted to the Royal Infirmary, Sunderland, on Feb. 5, 1936. She complained of blood in the urine for the last four months. The urine is stated to have been bright-red in the morning but became brown for the rest of the day. There was no frequency and no pain ordinarily, but she had had slight pain in the right groin during her periods for the last few months.

For about five years she has vomited during the two days preceding menstruation. Nothing was found on abdominal examination, and owing to the fact that she was menstruating and had tonsillitis she was discharged on Feb. 8.

On Feb. 25 she was re-admitted. The bladder was found normal at cystoscopy on Feb. 27, but bright-red urine was seen coming from the right ureter and normal urine from the left. The pyelogram was normal on both sides. Examination of ureteral catheter specimens showed normal urine from the left and red blood-cells only from the right. The catheter specimen of bladder urine was examined on Feb. 7, when it was found to have a few pus cells in addition to the above. The blood-count on March 16 was: hæmoglobin 56 per cent, red blood-cells 3,660,000 per c.mm., colour index 0.78, no white-cell abnormality. Operation was refused.

She was again admitted on April 3. There was no hæmaturia for two weeks. The urine was examined on April 6, when a few pus cells were present, but no pathogenic organisms. The right kidney was removed on the same day, when the fat over the pelvis was seen to be very hard and adherent, otherwise the kidney looked normal. She was discharged on April 21.

REPORT ON KIDNEY.—The kidney as a whole presented no macroscopic abnormality, but when the renal pelvis was inspected it showed some swelling of the mucous membrane, and there was a small uratic deposit in one of the calices. When this deposit was cleared away there was seen a minute red nodule about the size

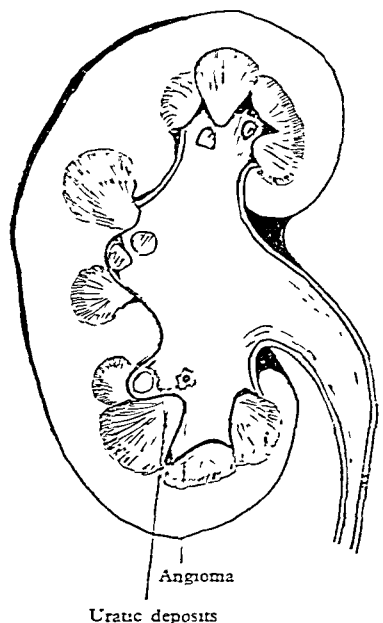


FIG. 291.—Diagrammatic view of the pelvis of the kidney to show the site of the angioma.

of a pin's head and evidence of recent bleeding therefrom (*Fig. 291*). Microscopical sections through the nodule showed what appeared to be an angioma (*Figs. 292, 293*).

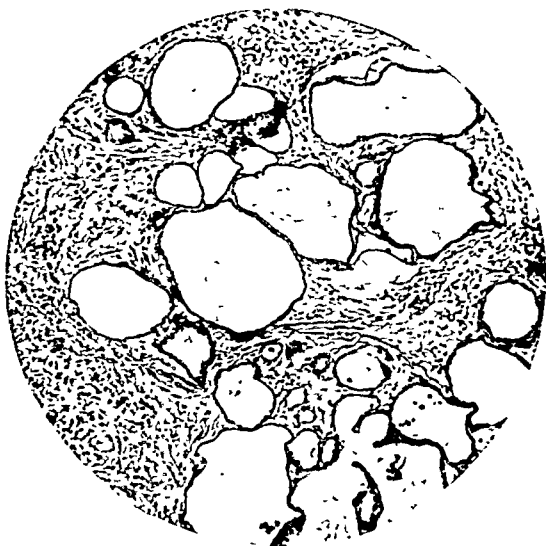


FIG. 292.—Microscopical section through the angioma. ($\times 120$.)



FIG. 293.—The same, more highly magnified. ($\times 275$.)

I have to thank Mr. H. Ross, Hon. Surgeon to Sunderland Royal Infirmary, under whose care this patient was, for the clinical notes of the case.

DIFFUSE CARCINOMATOUS INFILTRATION OF THE BONES OF THE FACE AND SKULL SIMULATING LEONTIASIS OSSEA

By ROBIN PILCHER

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WITH NOTES ON THE X-RAY EXAMINATION

By GWEN HILTON

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CLINICAL NOTES.—W. S., male, aged 56, was admitted to University College Hospital on Sept. 12, 1935, complaining of a painful swelling of the right side of the face for a year and a purulent discharge from the right nostril for a week. There was a history of treated syphilis since 1904, and in 1913 there were enlarged glands in the neck, said to have been tuberculous. There had been several attacks of ulceration of the throat, the last in 1922. A year before admission the teeth had been extracted, and according to the patient the swelling of the face first appeared shortly after this operation. The patient was sent to the Hospital by Mr. E. A. Peters, who regarded the condition as "malignant infiltration superimposed on syphilitic scarring".

The patient's general condition was poor, and he was in constant pain. The appearance of the face is shown in *Fig. 294*. The oedema of the eyelids was at first more extensive, but subsided during the first week in hospital. The skin around the right nostril and the right angle of the mouth was anæsthetic, but there was no facial weakness. The mouth was edentulous. There was intense leucoplakia of all parts of its mucous membrane, the change being most advanced in the palate and fauces, which were contracted and immobile. The concavity of the palate was almost obliterated by swelling, and the upper alveolus was uniformly expanded. No change was observed in the shape of the lower alveolus. On the right posterior end of the upper alveolus was a patch of superficial ulceration, but elsewhere the surface of the dense white mucous membrane was intact. The right side of the nose was blocked, and mucopus was running from the right nostril and down the nasopharynx. In the left posterior triangle two soft glands were palpable, and in the right posterior triangle was a small scar. The swelling of the face was apparently due partly to oedema of the soft tissues, but there was also enlargement of the underlying bone. Some thickening of the skull was palpable in the right frontal and temporal regions, the surface of the bone being slightly nodular. No clinical evidence of carcinoma was found in the mouth or nose. There was a slight secondary anæmia, and the Wassermann reaction of the blood was positive.

Anti-syphilitic treatment was started, and there was an immediate slight improvement which was not maintained. A piece of the right upper alveolus was excised for histological examination. The biopsy wound did not heal, but took on the appearance of a carcinomatous ulcer. The general condition deteriorated rapidly, and the patient died five weeks after admission.

X-RAY EXAMINATION (*Figs. 295, 296*).—Pathological changes are seen in the whole of the radiograph of the skull and the mandible, but are most marked in the maxillary and malar bones. The lesions in the vault are purely destructive, no new bone formation being seen. They appear as irregular osteoporotic areas scattered throughout the bones, giving the whole a worm-eaten appearance. They are chiefly localized in the frontal bones, but they are also present to a less extent

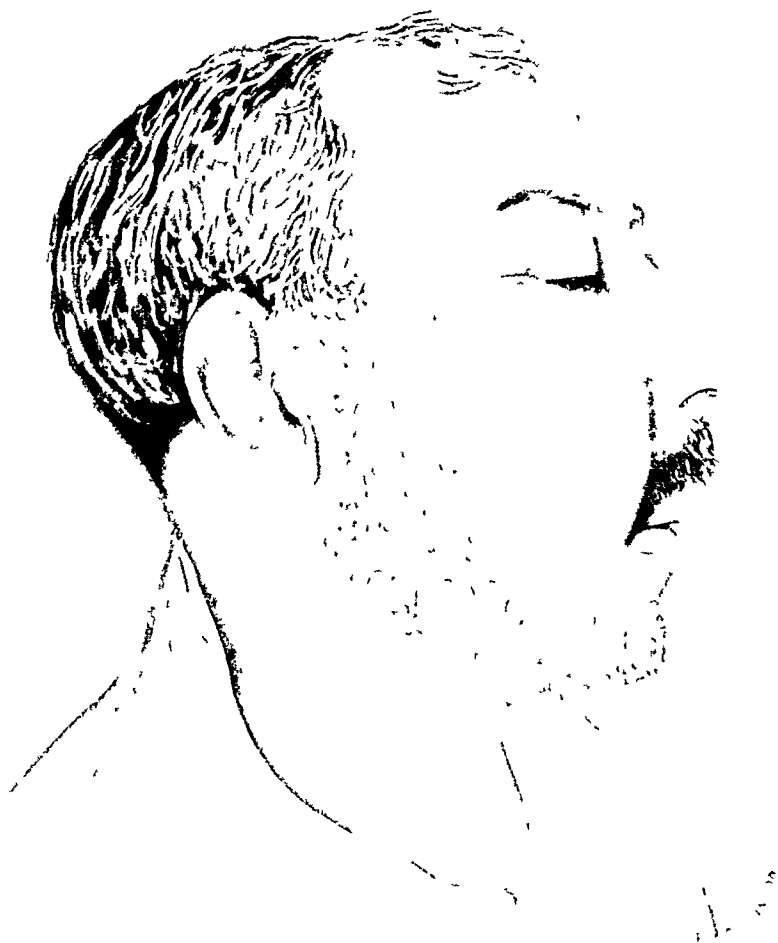


FIG 294—Appearance of the patient on admission.

in the parietal bones and in the squama of the temporal bones. Very few lesions are seen in the occipital bones. The inner and outer tables are not thickened anywhere and have smooth margins except in the frontal bone, where the outer table appears to be rarefied in several places. The floor of the anterior fossa is eroded, and the boundaries of the sella turcica are barely visible. The frontal, ethmoidal, and sphenoidal sinuses are obscured and their margins are impossible to define.

The changes in the bones of the face appear to be somewhat different. All normal contours have been lost. The bones have been replaced by a shapeless mass of spongy appearance with but slight calcification. This is so lacking in density that an antero-posterior view almost suggests that the lower part of the front of the skull is missing. The cervical spine is clearly visible through it. No indication of the position of the maxillary antra can be detected. The orbital fossæ are partially filled with the structureless material.

Osteoporotic areas are present in both mandibles, particularly in the ascending rami.

The total appearances in the vault resemble those of metastatic carcinoma, but the appearances in the face bones, which are the dominating features of the



FIG. 295.—Radiograph of the skull and mandible, lateral view.

FIG. 296.—Radiograph of the skull and mandible, antero-posterior view

radiograph, are similar to those described by various authors as one of the types of leontiasis ossia.

POST-MORTEM EXAMINATION.—A complete post-mortem examination was made, and no evidence of malignant disease except that in the mouth and the bones of the face and skull was found. The ulcer in the mouth had extended, and involved the right half of the palate. The upper alveolus, especially on the right side, was thickened and softened. A piece of the right maxilla including the alveolus and the floor of the antrum was easily cut out with a knife. There appeared to be no infiltration of the soft tissues of the cheek, and the cavity of the antrum was not obliterated. When the scalp was turned down it was again noted that the soft tissues were free of macroscopic signs of growth. The surface of the skull in the right frontal and temporal regions was nodular. The right orbit was explored and no growth was found in it. There were thickening and softening of the great



FIG. 297.—Microscopical section of maxilla. Epithelial pearls in fibrous stroma. In other parts of this section the stroma is scanty. ($\times 84$.)



FIG. 298.—Microscopical section of frontal bone. Islets of carcinoma surrounded by fibrous tissue. ($\times 84$.)

wing of the sphenoid, but it could not be cut as easily as the maxilla. In addition to the maxilla, pieces of bone were removed for histological examination from the right sphenoid, temporal, and frontal bones. The striking feature of the malignant process was its apparent localization to bone.

Histology.—The appearances of the biopsy and post-mortem specimens are similar. In all of them there is invasion and destruction of bone by well-differentiated squamous carcinoma with cell-nest formation. Where the growth has invaded the bone there is a fibrous-tissue reaction and some new bone formation (Figs. 297, 298). Except in the maxilla the fibrous tissue is in excess of the carcinoma, but in none of the sections examined was fibrous-tissue replacement of bone found without carcinoma. The part of the antrum included in the section of maxilla has a normal columnar epithelial lining.

Comment.—On admission this was thought to be a case of leontiasis ossea. Carcinoma was not suspected until biopsy was performed. The site of origin of the carcinoma was not discovered, but was probably one of the tooth sockets of the upper jaw. Before the biopsy there was nothing in the mouth to suggest carcinoma, and at post-mortem the cavity of the antrum was not obliterated and its lining was found to be normal columnar epithelium. When the result of the biopsy was known there was still doubt as to the nature of the diffuse change seen in the radiographs. It was thought that there might be two conditions present, carcinoma and osteitis fibrosa, but no evidence of the latter was found post mortem. The unusual feature of the case is the local spread of carcinoma in the bones rather than in the soft tissues, which showed no macroscopic involvement at post-mortem.

EXTREME BENIGN ENLARGEMENT OF THE PROSTATE

By J. McFADZEAN

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AND W. E. COOKE

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FROM time to time prostatic tumours are reported on account of their size. The remarkable dimensions and weight of the prostate in the present case may not constitute a record, but it is unique in our experience and in the experience of many surgeons and pathologists who have seen it.

The patient, aged 71 years, was admitted to hospital for acute retention of urine. An attempt to pass a soft rubber catheter had been made at home but had failed on account of excessive hæmorrhage. A suprapubic cystostomy was performed immediately and a large-bore de Pezzer's catheter inserted, the bladder wall first being pierced with a trocar whose cannula was just large enough to carry the stretched catheter. The urine was released at the rate of 2 oz. every hour. Drainage was continued for a fortnight, when another attempt was made to pass a catheter per urethram. This again resulted in copious hæmorrhage.

The non-protein nitrogen was 35.5 mg. per 100 c.c. of blood. Suprapubic prostatectomy was decided upon and performed under a spinal anæsthetic. On

opening the bladder the prostate was found to occupy almost the whole of the cavity. Enucleation was difficult on account of the large size of the growth, the upper surface of which came in contact with the knuckles and prevented the base of the gland being reached with the finger. (We may mention that J. McF. has exceptionally long fingers.) Finally the gland was removed by a process of fragmentation and with it two horned concretions. The patient made an uneventful recovery.

The pieces of gland were weighed immediately after the operation, and turned the scale at 1 lb. 6½ oz. The larger pieces were sutured together to obtain a photographic record. Some idea of the size will be gained from the photograph (Fig. 299), but large portions are missing. The tumour, as seen in the photograph,

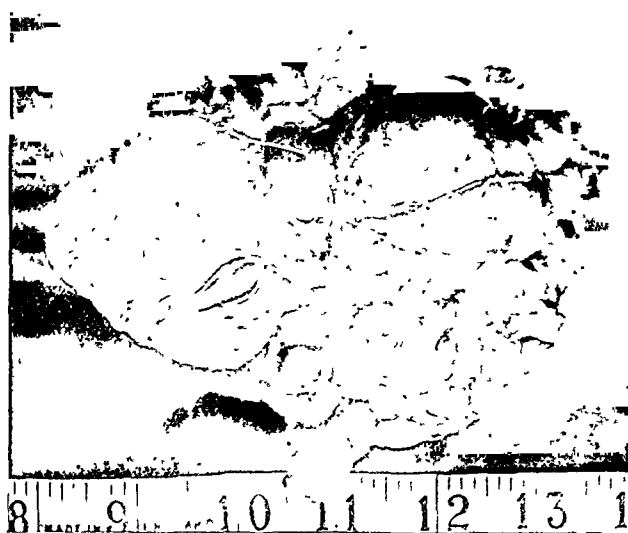


FIG. 299.—Photograph showing the larger pieces of the removed prostate sutured together to give an indication of the extraordinary size of the tumour. ($\times \frac{1}{2}$.)

after being in 10 per cent formalin for two months, measured 6 in. by 5¼ in. by 4 in. in thickness. Many of the small rounded masses seen in the photograph were attached to the main body of the tumour by narrow pedicles and became readily detached from it.

HISTOLOGY.—All the tissues of the prostate subscribe to the enlargement of the gland and although one might be inclined to think the glandular elements preponderate and term the condition an adenoma, examination of many sections shows increase in the fibrous and muscular elements to an equal extent. Perhaps the correct category to place the tumour would be in the simple senile nodular hypertrophy class as suggested by Kettle.¹

REFERENCE

- ¹ KETTLE, E. H., *The Pathology of Tumours*, 2nd ed., 252. London.

REVIEWS AND NOTICES OF BOOKS

A Textbook of Surgery. By American Authors. Edited by FREDERICK CHRISTOPHER, B.S., M.D., F.A.C.S., Associate Professor of Surgery at Northwestern University Medical School, etc. Royal 8vo. Pp. 1608-xix, with 729 illustrations. 1936. Philadelphia and London: W. B. Saunders Co. 42s.

THIS *Textbook of Surgery*, in one volume, is unique inasmuch as the contributors include surgeons of the highest distinction in America and each branch of surgery is dealt with by a recognized specialist—such names as Adson, Smith-Petersen, Balfour, Judd, and Evarts Graham may be mentioned.

The book is designed for students, and, although it would be rather heavy reading for such in the early stages of their careers, it should be of great value both to those just about to qualify and to post-graduates. As is natural in such a work there is some inequality in the sections: for example, those dealing with the Breast, Thorax, and Abdomen are admirable, whereas Tuberculosis of Joints is dealt with very briefly and in a manner which gives little help to the student.

There are valuable sections on Roentgenology and Aseptic Surgical Technic, and on such important matters as the management of the surgical diabetic patient and the care of patients after operation.

The volume is beautifully printed in clear type, and the illustrations, which are very numerous, are on the whole admirable.

A single volume text-book of this type has been badly needed, and Dr. Christopher's great work should amply fill this want.

The Operations of Surgery. By R. P. ROWLANDS, M.S. (Lond.), F.R.C.S., late Surgeon to Guy's Hospital, etc.; and PHILIP TURNER, B.Sc., M.S. (Lond.), F.R.C.S., Consulting Surgeon to Guy's Hospital, etc. Eighth edition. Volume I: The Upper Extremity. The Head and Neck. The Thorax. The Lower Extremity. The Vertebral Column. Royal 8vo. Pp. 1405+x, with 435 illustrations (38 in colour). 1936. London: J. & A. Churchill Ltd. 36s. net.

THIS book is the direct descendant of Jacobson's *Operations of Surgery*, a volume which attained a unique place in surgical literature. In its subsequent editions it has maintained the characters which led to its high repute. It has always been a reliable guide to the younger practising surgeon, informing him what it is possible to achieve under average circumstances, warning him of dangers and accidents which lurk unsuspected, and acquainting him with the results which in the end may ensue.

The absolute dependability of the advice in its pages, the freedom from exaggeration, the balanced judgement of its presentation, were what made it of such unusual value to the junior, and even to the senior, surgeon. Mr. Turner, in bringing out this new edition, has kept closely to the ideals of its predecessors. Mr. R. P. Rowlands, before his death, had prepared much material for this edition, but those sections which he left uncompleted have been placed in the hands of Mr. W. H. Ogilvie, Mr. Grant Massie, and Mr. A. R. Thomson, whilst the gynæcology has been largely re-written by Mr. G. F. Gibbard. Capable as these hands are we cannot but be aware of the danger in multiple authorship of the book losing some of its distinctive character, in which its real value lies. One hesitates to accept it as inevitable that modern specialization should make multiple authorship necessary. However, the book does not seem to have suffered much in this respect in *Volume I*. It is still the safe guide for the junior surgeon, pleasantly written and excellently illustrated. All the usual operations performed by the general surgeon are well and clearly described, and there

are accounts of some useful procedures, such as Hey Groves's method for recurrent dislocation of the shoulder, which are rarely found elsewhere. A few not very important criticisms may be made. In the treatment of trigeminal neuralgia only one route for reaching the Gasserian ganglion for injection is mentioned; there is a long description of removal of the ganglion, whilst the usual division of the root is relegated to small print; and no account of the intracranial operation of division through the occipital region is given. Facial-accessory anastomosis is surely an operation which might have been omitted, and so perhaps is Lane's operation for cleft palate. The description of removal of a part of the lung is poor, and we should have liked to see more details given of how to perform Lorenz's useful bifurcation operation. In *Fig. 125* the artist has depicted the humerus with the head pointing the wrong way.

We have been led to expect such a high standard in this book that a reviewer is apt to exalt minor blemishes to the position of subjects for serious criticism. There is in reality very little matter of the latter nature to be found, and the book remains one of the best guides to operative surgery in the English language. The author is to be heartily congratulated on the publication of this, the eighth and best, edition which has yet appeared.

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London, etc. Second edition. Demy 8vo. Pp. 842 + x, with 812 illustrations. 1936. Bristol: John Wright & Sons Ltd. 50s. net.

THE first edition of this text-book was issued in two volumes, the first of which was reviewed in this JOURNAL in April, 1931, and the second in April, 1932; in this, the second edition, these have been combined into one volume, the total number of pages being increased from 795 to 842. Certain portions have been re-written and the whole thoroughly revised. Mr. Eric Watson-Williams is again responsible for the sections upon the nose, ear, and larynx, and upon intracranial suppuration, while Mr. Humphrey Neame deals with the eye and orbit.

The very favourable opinion we expressed of the first edition has been decidedly enhanced, and we can recommend this work not only to the 'comparatively isolated' and occasional surgeon for whom, primarily, it was written, but also to others of much wider experience. There are such excellent accounts not only of the emergency operations themselves, but of their complications and the best measures of meeting them, that it is obvious that the author has had an exceptionally extensive and varied practical acquaintance with the surgery of emergencies.

The book is splendidly illustrated, clearly printed on very good paper, and thoroughly indexed.

Neurological Surgery. By LOYAL DAVIS, M.S., M.D., Ph.D., D.Sc. (Hon.), Professor of Surgery and Chairman of the Division of Surgery, Northwestern University Medical School, Chicago, Illinois. 5½ × 9½ in. Pp. 429, with 172 illustrations and 2 plates. 1936. London: Henry Kimpton. 28s. net.

THE field of neurological surgery has expanded considerably during the last twenty years, and yet there is still a feeling amongst some medical men that the surgery of the brain is practically useless. It is because of this attitude that Loyal Davis was prompted to write this book on neuro-surgery. The book is not written for the neuro-surgeon but for the general surgeon who may have to undertake the surgery of the brain; it will also be of use to the general practitioner in convincing him that success often follows surgical operations on the central nervous system.

The first chapter is devoted to a lucid account of the way in which a neurological diagnosis may be obtained. Cranio-cerebral injuries are carefully considered, and the modern tendency of non-interference, unless there are definite signs, is stressed. The largest chapter in the book is devoted to intracranial tumours, and a well-balanced account of the various types is given. The illustrations on the whole are excellent, especially the microphotographs. Intracranial abscesses receive scant attention. The spinal cord, the peripheral nerves, and the autonomic nervous system are dealt with in separate chapters.

Although the literature dealing with neurological surgery is becoming voluminous, yet we feel that there is room for this book, especially for senior students who want a practical monograph on the subject. It is a great pity that no bibliography is incorporated in the book; a few scattered references at the foot of some pages is not quite in keeping with a monograph dealing with a special subject.

Cystoscopy and Urography. By JAS. B. MACALPINE, F.R.C.S., Hon. Surgeon and Surgeon in Charge of the Genito-urinary Department, Salford Royal Hospital, Manchester. Second edition. Large 8vo. Pp. 478 + xv, with 297 illustrations and 14 coloured plates. Bristol: John Wright & Sons Ltd. 30s. net.

THE first edition of this book appeared nine years ago: at that time we ventured to predict a great success for it as one of the best surgical monographs which have been published during the last thirty years.

The rapidly increasing part played by urography since the introduction of uroselectan has induced the author to alter the title of his book from *Cystoscopy* to the present *Cystoscopy and Urography*, and has made it necessary for him to remodel some chapters and to add an entirely new section.

The book is admirably turned out; it is light to hold and of convenient size; the printing is clear and easy to read; the many illustrations are strictly relevant to the text, and both these and the coloured plates are of real artistic merit. Last, but not least, the book is written in such a clear and concise style that it is a pleasure to read, and one feels that the author is a master of his subject; he has not only had an extensive experience but has read widely and thought deeply about genito-urinary surgery.

The cystoscopic pictures of the bladder are really superb, and their value is much increased by the simple device of giving a linear indication in the margin of the direction in which the beak of the cystoscope was pointing; this is an improvement that will be greatly appreciated by all cystoscopists.

The sections on perurethral prostatectomy, on pelvic resorption, and on urography are all excellent, and are good examples of the author's balanced judgement.

A new chapter is devoted to the consideration of that very important subject—the congenital abnormalities of the kidney and ureter: it is clear and thoroughly up to date, the one exception that might be taken to it is that in the description of hypoplasia of the kidney no mention is made of the absence of the usual number of papillæ; unless this deficiency is taken into account, the condition is difficult, or impossible, to distinguish from atrophy.

A most interesting chapter is devoted to what is often called Schramm's phenomenon, though wrongly according to Macalpine. This is a condition, well recognized by all cystoscopists, in which the posterior wall of the prostatic urethra and the verumontanum can be clearly seen on withdrawing the cystoscope from the bladder, it is met with in men, and is nearly always associated with disease of the nervous system. The author has christened it 'funnel-neck deformity of the bladder', and he goes so far as to say that no cystoscopy in the male should be considered complete until the cystoscope has been withdrawn into the prostatic urethra; this is indicated even more imperatively in the presence of signs of obstruction to micturition and of trabeculation of the bladder. That it is a sign of real value is proved by the records of cases in which the discovery of this phenomenon has antedated the finding of other signs of nervous disease. Whilst there can be no doubt of the existence of this deformity, its mechanism is by no means clear, cystograms taken in such cases usually reveal no leakage of urine into the dilated prostatic urethra, and it has been suggested that the weight of the beak of the cystoscope may depress the posterior wall of the internal meatus.

In the section on Urography the author makes a suggestion that may prove of considerable value in the future, instead of estimating the amount of residual urine by catheterizing a case of enlarged prostate, he thinks it would be feasible to do so by taking a skiagram of the bladder after the injection of uroselectan—of course, after the patient has tried to empty his bladder. This, if successful, may save much heartburning to the surgeon and may add many years to the patient's life.

Another ingenious suggestion is that of giving a spinal anæsthetic to the patient suffering from a stone in the ureter; this might induce a relaxation of the ureteric muscle analogous to the relaxation obtained in cases of intestinal obstruction.

There is a good index and a delightful absence of misprints; we warmly recommend this monograph to our readers and we offer our congratulations to the author, the artist, and the publishers; they have produced a book which is a credit to British surgery.

A Handbook of Urology for Students and Practitioners. By VERNON PENNELL, M.A., M.B., B.Chir. (Cantab.), F.R.C.S. (Eng.), Hon. Surgeon and Surgeon with Charge of Urological Department, Addenbrooke's Hospital, Cambridge; etc. Cr. 8vo. Pp. 224 + viii, with 34 illustrations. 1936. London: Cambridge University Press. 7s. 6d.

IN this work the author's aim is to give a short and concise account of urinary diseases, with their method of investigation and treatment. He is a urologist of some experience, for he worked with Mr. Canny Ryall at the All Saints' Hospital, London, and is now in charge of the Urological Department at Addenbrooke's Hospital, Cambridge.

Mr. Pennell has a light touch and the gift of making his subject interesting; the sections on growths of the bladder and on the treatment of the enlarged prostate are very good, but the pleasure derived from reading this book is spoilt by defects which, if minor, are undoubtedly too numerous.

To give a few instances: in the section on tests of renal efficiency, phenol red is recommended and stated to be the substance most commonly used for this purpose—though we doubt whether this is so, even in the United States where it originated—and yet, after reading this, we are astonished to find that this test is never mentioned again; indeed, whenever a test of renal function is indicated, the author invariably uses indigo-carmin.

In the discussion on the treatment of pyelitis it is said that a ketogenic diet may be worthy of a trial, "although as a rule it is not well borne"; with this we agree, but are not a little surprised to find that no mention is made of mandelic acid; we are still more surprised to find, in the appendix of drugs at the end of the book, a prescription for administering this new and efficacious drug.

We consider his assertion that the development of an intestino-vesical fistula is generally due to malignant disease to be inaccurate; nearly fifty years ago Harrison Cripps wrote an admirable monograph on this very subject, in which he pointed out that a commoner cause was inflammation of the colon; this has been abundantly confirmed since, and most, if not quite all, urologists would agree that the majority of such cases of pneumaturia are due to diverticulitis.

Though Mr. Pennell to some extent disarms criticism by confessing that he is acutely conscious of the deficiencies in his book, we feel that he should have taken more care to exclude any inaccuracies in a book destined for students.

Again, we think that no work on urology can be considered up to date which advises for the removal of a stone from the lower end of the ureter a curved incision similar to that for the extraperitoneal ligature of the common iliac artery; surely, surgeons agree that the median subumbilical incision is infinitely preferable—this is not even mentioned.

The author's grammar and spelling are sometimes strikingly unconventional, or perhaps it would be more charitable to assume that he had no chance of reading over the proof sheets. We cannot congratulate him on his coloured plates; the cystoscopic view of a papilloma of the bladder (*Fig. 23*) is very much below the standard of modern cystoscopic atlases.

The Principles and Practice of Urology. By FRANK HINMAN, A.B., M.D., Clinical Professor of Urology at the University of California Medical School. Large 8vo. Pp. 1111, with 513 illustrations. 1935. London and Philadelphia: W. B. Saunders Company. 45s. net.

AFTER skimming over the eleven hundred pages of this vast work (no inconsiderable task: it would take many months to read it through as carefully as it deserves), two adjectives rise instinctively to the surface; they are *astounding* and *encyclopaedic*.

Dr. Frank Hinman is well known on this side of the Atlantic for his always interesting and often highly original work on several subjects; to mention only two—his researches on

experimental hydronephrosis and renal counterbalance. In addition to this, he gives an autobiography of well over one hundred articles, and it seems to us astounding that he has found time to collect all the information set out in this volume.

The work is encyclopædic, for in it will be found not only an account of genito-urinary surgery but sections on the anatomy and physiology of the organs concerned, their development and their evolution from the amœba up to man. In addition to all this, there are full accounts of the various methods of examination of these organs.

The author deserves the greatest credit for his industry, for he has not merely transposed the work of others, but he has assimilated this material and made it very definitely his own.

It is obvious that Dr. Hinman is an indefatigable collector, and this volume must take its place in the library of every urologist if only for the numerous and excellent illustrations and the many rare and curious cases he has gathered together.

Naturally, the best sections are those on subjects at which he has worked: pyelovenous backflow, hydronephrosis, and renal counterbalance; the last is particularly convincing, and, in our opinion, explains the pessimism of the French urologists as to the effect of plastic operations on the ureter on renal function.

The work has one obvious defect: so much attention is devoted to pathology and treatment that diagnosis sometimes suffers a partial eclipse. The author has been so anxious to make his book include every possible scrap of information available that the effect is occasionally rather overwhelming; one sighs for the clinical pictures drawn by the older authors—they were more artistic if less complete.

We are surprised to find in the article on tuberculosis of the kidney that no mention is made of the fact that in the female the genital organs escape infection, in such marked contrast to the course of the disease in the male.

Nor does he refer to the possibility of feeling the thickened, tuberculous ureter through the vagina or the rectum; this is a very valuable physical sign, for it can be detected by the general practitioner; we cannot find any reference to this method of diagnosing a stone in the lower end of the ureter.

An even more surprising omission is that of any description of Schramm's phenomenon—funnel-neck deformity of the bladder; particularly as there is an otherwise excellent discussion on the pathology of the bladder in diseases of the nervous system.

We point out these defects in no spirit of hostile criticism but to enable Dr. Hinman to rectify them in his second edition, which will surely be called for later.

Personally, we should omit the diagrams in any future edition; whilst fully agreeing with the author as to the value of diagrams in teaching, we look on them as useful chiefly for simplifying a subject; the diagrams in this volume are so complicated that it is quite impossible to understand them.

We heartily commend this work to our readers, but we consider it suitable only for surgeons who wish to keep it for reference; we think that the student and the general practitioner would get an altogether erroneous impression from it of the diagnosis of genito-urinary disease.

La Pratique Chirurgicale Illustrée. Fascicule XX. Large 8vo. Pp. 259, with 219 illustrations. 1936. Paris: G. Doin et Cie. Fr. 70.

In this volume Victor Pauchet writes on gastrectomy for duodenal ulcer and partial gastrectomy for large ulcers of the stomach, in association with Gabriel Luquet; on the cure of transverse colostomy by an operation entitled ileo-colo-rectostomy; and on common duct obstruction, in association with Dr. P. L. Gac.

The remaining articles are by various writers on operations for cleft palate, œsophageal diverticula, sublingual cyst, extravasation of urine, cancer of the urethra, cystotomy, and a new and ingenious type of Einhorn tube designed to prevent rotation in the stomach before passage into the duodenum.

The excellent illustrations of S. Dupret enable the reader to follow the details of the operations with only an occasional reference to the text, and the same high standard of the previous volumes has been maintained.

The gastric and duodenal operations here described have been presented in previous volumes with minor modifications and leave nothing to be desired.

The method described to repair the continuity of the intestinal tract after a colostomy on the left side of the transverse colon is very ingenious and was described by Pauchet at the subsection of Proctology at the Royal Society of Medicine some years ago. In the case here described a colostomy had been performed some years before for ulcerative colitis, which had produced an extensive stricture of the descending colon.

The article on the surgery of the common duct when obstructed is excellent and very well illustrated.

At the end of the volume there is an index relating to the twenty volumes now published.

Orthopædic Surgery. By WALTER MERCER, M.B., Ch.B., F.R.C.S.(Edin.), F.R.S.(Edin.), Assistant Surgeon, Royal Infirmary, Edinburgh, etc. With a Foreword by JOHN FRASER, M.C., M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh. Second edition. Medium 8vo. Pp. 906 + xi, with 408 illustrations. 1936. London: Edward Arnold & Co. 40s. net.

THE appearance of a second edition of this work within four years of its first publication indicates that it has been appreciated, and the enlargement of the volume by two hundred pages and the extensive revision shows that the author has been industrious and anxious to perfect it and bring it up to date. In this he has been so successful that it may now be considered to be one of the most complete general accounts of orthopædic surgery to be found in the English language. The book is suitable for students, for those reading for higher examinations, and as a work of reference.

Studies on the Healing of Fractures with Special Reference to the Significance of the Vitamin Content of the Diet. By JOHN HERTZ, M.D., Assistant Surgeon, Department C, Surgical University Hospital, Copenhagen. Super royal 8vo. Pp. 286, with 82 illustrations. 1936. Copenhagen: Levin & Munksgaard. (London: Oxford University Press.) Kr. 15.

DR. HERTZ has made a review of the literature and carried out an extensive series of experiments upon animals, testing the effects of deficiency in the various vitamins upon both the inflammatory reaction which follows the trauma immediately and upon the later stages of healing.

The experiments are well presented and very beautifully illustrated. In one of the earlier chapters the author adopts the Hansen-Hägqvist syncytial theory of cellular growth in osseous tissues. It is somewhat remarkable that in an otherwise very complete bibliography references to these two authors are omitted.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. Second edition. Fcap 8vo. In two volumes. Pp. 180 + viii, with 37 illustrations. 1936. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each net.

ONE of a series of small monographs giving a compact synopsis of the treatment of fractures. The detail is scarcely sufficient to enable the reader to treat any but the simplest fracture himself, but it is sufficient for revision by students before examination or for practitioners.

The Foot. By NORMAN C. LAKE, M.D., M.S. (Lond.), F.R.C.S., Senior Surgeon and Lecturer on Surgery, Charing Cross Hospital. Large 8vo. Pp. 330 + vii, with 95 illustrations. 1935. London: Baillière, Tindall & Cox. 12s. 6d. net.

MR. LAKE has written an account of the normal foot, its evolution and development, functions, and minor variations and abnormalities. He does not include talipes or paralytic deformities, and he gives only a very brief account of injuries. In the preface he calls attention to the fact that trivial lesions of the feet cause much disability and receive scant attention in medical teaching, a complaint that is perhaps less true now that students are obliged to receive instruction in orthopædics. But this book certainly serves to fill a gap, and gives much information that is useful to the medical student and practitioner. It is also written so as to appeal to the chiropodist and masseuse.

In general the matter is simply expressed though adequate; but when operative measures are mentioned the advice given is apt to be tentative and uncertain, a matter of some importance in that the general practitioner or the masseuse or chiropodist is left with the feeling

that operations on hammer-toes or bunions cannot be safely suggested. Another point of criticism is that in a work which deals with the foot largely from the point of view of function, one might expect to find more detail of exercises for re-education in proper methods of standing and walking.

Le Cancer (Etudes Anatomo-Cliniques).—By I. STOIA, Docent de la Faculté du Médecine de Bucarest; Médecin chef du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc.; and P. STANCIULESCU, Assistant du Laboratoire central d'Anatomie-Pathologique de l'Hôpital Brancovan; etc. With a Preface by Professor CH. LENORMANT. Super royal 8vo. Pp. 322, with 147 illustrations. Paris: Masson et Cie. Fr. 55.

THIS book consists of ten short monographs on different forms of cancer in which a study has been made of the relations of the clinical symptoms to the anatomical and pathological appearances of the tumour material.

The Life and Works of Charles Barrett Lockwood (1856-1914). By ERIC C. O. JEWSBURY, M.A., B.M., B.Ch. (Oxon.). The Wix Prize Essay, St. Bartholomew's Hospital, 1934. Crown 8vo. Pp. 104 - viii, with 12 illustrations 1936. London: H. K. Lewis & Co., Ltd. Cloth, 3s. 6d. net; paper covers, 2s. 6d. net.

ANYBODY familiar with the surgical side of Bart's thirty years ago will recollect what an outstanding position Lockwood occupied at that time. The admirable account of his life and work, which has been written by Eric Jewsbury, is particularly successful in that it pictures the man as we knew him with all his characteristics and eccentricities.

This little biography has been penned without fear, and it shows very well why so many people were repelled by Lockwood, if they did not frankly dislike him. But while this side of his character has been justly portrayed, it is made obvious that these characteristics were merely a cloak for a sensitive and tender nature, very apt to be misunderstood.

Lockwood was a great worker and writer, and the author has done well to supply in an appendix an apt survey of the outstanding publications for which this surgeon was responsible. In addition there is a complete bibliography, a list of authorities, the names of those to whom he has been indebted for much information, and, as though that were not sufficient, some incidental references that have a bearing on the matter in hand. It is seldom indeed that a biography so complete and so successful has been presented in such a small compass.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Text-book of Pathology. By Sir ROBERT MUIR, M.A., M.D., Sc.D., LL.D., F.R.S., Professor of Pathology, University of Glasgow; Pathologist to the Western Infirmary, Glasgow. Fourth edition. Medium 8vo. Pp. 994 - viii, with 571 illustrations. 1936. London: Edward Arnold & Co. 35s. net.

Appendicitis: When and How to Operate. By W. J. STEWART MCKAY, M.B., B.Ch., B.Sc., Senior Surgeon, Lewisham Hospital, Sydney. Demy 8vo. Pp. 260 - xii, with 16 illustrations. 1936. Sydney: Angus & Robertson Ltd. 12s. 6d. net.

Die Wirbelgelenke. By Prof. Dr. MAX LANGE, Director of the Orthopædic Clinic, Munich. Second enlarged edition. Large 8vo. Pp. 138 - viii, with 94 illustrations. 1936. Stuttgart: Ferdinand Enke. RM. 10.

Techniques Chirurgicales. By A. GOSSET, Chirurgien de la Salpêtrière, Professeur de Clinique chirurgicale à la Faculté de Médecine de Paris; with the collaboration of MM. L.-G. AMIOT, IVAN BERTRAND, JEAN CHARRIER, P. FUNCK-BRENTANO, J. GARCIA-CALDERON, JEAN GOSSET, P. HAUDUROY, R. LEDOUX-LEBARD, R. LEBOVICI, G. LEWY, P. PETIT-DUTAILLIS, P. ROUCHÉ, R. SAUVAGE, G. SEILLÉ, R. SOUPAULT, M. THALHEIMER, and E. WALLON. Imperial 8vo. Pp. 434 - viii, with 219 illustrations. 1936. Paris: Masson et Cie. Paper covers, Fr. 105; bound, Fr. 125.

- The Surgical Technic of Abdominal Operations.** By JULIUS L. SPIVACK, M.D., Assistant Professor of Surgery, University of Illinois College of Medicine; Professor of Operative Surgery and Surgical Anatomy, Cook County Graduate School of Medicine; etc. $10\frac{1}{2} \times 7\frac{1}{2}$. Pp. 718 + xvi, with 362 illustrations. 1936. Chicago: S. B. Debour.
- Minor Surgery.** By FREDERICK CHRISTOPHER, S.B., M.D., F.A.C.S., Associate Professor of Surgery at the Northwestern University Medical School, Chicago; Chief Surgeon at the Evanston (Ill.) Hospital. With a Foreword by ALLEN B. KANAVEL, M.D., F.A.C.S., Professor of Surgery at the Northwestern University Medical School. Third edition, reset. Large 8vo. Pp. 1030, with 709 illustrations. 1936. London and Philadelphia: W. B. Saunders Co. Ltd. 42s. net.
- Vorträge aus der praktischen Chirurgie.** Edited by ERICH LEXER. Heft 8. Die Kurzwellenbehandlung in der Chirurgie. By Dr. ALFONS LOB, X-ray Department, University Surgical Clinic, Munich. Large royal 8vo. Pp. 68. 1936. Stuttgart: Ferdinand Enke. R.M. 3.60.
- The Relief of Pain. A Handbook of Modern Analgesia.** By HAROLD BALME, M.D., (Durh.), F.R.C.S. (Eng.), D.P.H. (Lond.), Formerly Professor of Surgery and Dean of the School of Medicine, Cheeloo University, China. With an introduction by Sir E. FARQUHAR BUZZARD, Bt., K.C.V.O., LL.D. (Man.), M.D. (Oxon.), F.R.C.P. Large post 8vo. Pp. 392 + xvi. 1936. London: J. & A. Churchill Ltd. 12s. 6d. net.
- Recent Advances in Genito-urinary Surgery.** By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital; Surgeon and Urologist, Essex County Council; and NORMAN M. MATHESON, M.B., F.R.C.S., M.R.C.P., Surgeon, Central Middlesex County Hospital. Large post 8vo. Pp. 213 + viii, with 89 illustrations. 1936. London: J. & A. Churchill Ltd. 15s. net.
- Behandlung der Verletzungen und Eiterungen an Fingern und Hand.** By Professor Dr. M. ZUR VERTH, Privatdozent in the University of Hamburg. Second edition. $5\frac{1}{4} \times 8\frac{1}{2}$. Pp. 164 + viii, with 59 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 8.70; bound, RM. 9.60.
- Diseases of the Testicle.** By HAMILTON BAILEY, F.R.C.S., Surgeon, Northern Hospital; Surgeon and Urologist, Essex County Council; etc. Medium 8vo. Pp. 160 + viii, with 129 illustrations, of which 10 are coloured. 1936. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.
- The Patient Looks at the Hospital.** By FLORENCE G. FIDLER. With a Foreword by JOAN MALLESON, M.B., B.S. Crown 8vo. Pp. 128 + xii. 1936. London: Robert Hale & Co. 2s. 6d. net.
- Paget's Disease of the Nipple.** By KEITH INGLIS, M.D., Ch.M. (Sydney), Professor of Pathology in the University of Sydney; Formerly Director of the Kanematsu Memorial Institute of Pathology, Sydney Hospital. Crown 4to. Pp. 233 + xii, with 237 illustrations. 1936. London: Oxford University Press. 36s. net.
- Clinical Handbook for Residents, Nurses, and Students.** Being the Routine Methods of St. Vincent's Hospital, Sydney. By Members of the Staff of the Hospital. Edited by VICTOR M. COPPLESON, Ch.M., F.R.C.S., F.R.A.C.S., and DOUGLAS MILLER, Ch.M., F.R.C.S., F.R.A.C.S. Cr. 8vo. Pp. 205 + x. 1936. London and Sydney: Angus & Robertson Ltd. 6s.
- The Thyroid: Surgery, Syndromes, Treatment.** By E. P. SLOAN, M.D. Edited by Members of the Sloan Clinic. With a Foreword by WM. SEAMAN BAINBRIDGE, M.D. $10\frac{1}{4} \times 7\frac{1}{2}$. Pp. 475 + ix, with 99 illustrations. 1936. London: Baillière, Tindall & Cox. 45s.
- Surgery for Dental Students.** By PHILIP H. MITCHINER, M.D., F.R.C.S., C. E. SHATTOCK, M.D., F.R.C.S., E. G. SLESINGER, B.Sc., F.R.C.S., and CECIL P. G. WAKELEY, D.Sc., F.R.C.S. 9×6 . Pp. 364 + ix, with 105 illustrations. 1936. London: Baillière, Tindall & Cox. 12s. 6d.

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, K.B.E., LONDON

XII. TWO LIVERPOOL SURGEONS II. EDWARD ALANSON, A PIONEER IN HOSPITAL SANITATION

EDWARD ALANSON, Surgeon to the Liverpool Infirmary, was far in advance of his time. Overcrowded hospitals, badly situated, and with little or no sanitation, had not yet attained the dreadful notoriety to which they were destined. Liverpool Infirmary perhaps would in any case have escaped the appalling mortality of some of the other general hospitals, for it was near the sea and the medical students were few. Nevertheless, Alanson foresaw the dangers, and made a series of remarkable recommendations, which he published in his *Practical Observations on Amputation and the After-Treatment* in 1779. He says :—

“ The air in which the cure is to be conducted is a point worthy of your greatest attention ; if possible the room should be spacious and in an open wholesome situation. It is well known that in hospitals which are situated in populous towns and much crowded the salutary influence of the air is so altered that compound fractures and other important surgical cases prove peculiarly fatal ; and that amputations done in the country will be followed almost certainly with a speedy cure. The consequent symptoms are trifling, nearly the whole internal surface of the wound unites by the first intention ; the suppuration consequently is small and as soon as laudable, the secondary union takes place and the whole cure is speedily completed.

“ Many hospitals are so tainted by unwholesome effluvia that they are rather a pest than a relief to the objects they contain.

“ The following regulations are humbly recommended to the consideration of those who have the care of hospitals in want of such attention.

“ 1. No ward should be inhabited for more than the space of four months together ; for it is impossible to keep a room healthy, that is constantly crowded with diseased people ; the walls should be scraped, whitewashed and every other necessary means used for the purification of the air before the readmission of patients.

“ 2. The bed-stocks should be made of iron to prevent the lodgement of vermin and the more easy absorption of putrid matter.

"3. The bedding should be more frequently changed than is usually done ; and the bed-tick stuffed with chaff, hay, cut straw, or materials of such easy expence as to admit of their being frequently changed.

"4. Where a hospital is conveniently situated for the purpose all the patients that are able should carry out their bedding and expose it in the open air for several hours every day when the weather will permit.

"5. On the days of admission those patients that have inhabited foul ships, jails, cellars or garrets, workhouses or other infected places ; or whose clothes are dirty or suspected to contain vermin, before they are suffered to appear in the ward, should first be stripped and washed in the warm bath, and afterwards clothed with proper dresses, provided at the expence of the charity ; by which means the evil of importing infection, so detrimental to the salubrity of every hospital, would be greatly remedied.

"6. The dresses for the men may consist, chiefly of a clean shirt, jacket and trousers ; for the women, a shift, petticoat and bed-gown ; the rest may be supplied from their own clothing, which will easily admit of being first well cleaned.

"7. The infected clothes should be baked in an oven constructed for the purpose ; by which all vermin and infection will be destroyed ; and the clothes may be returned clean to the patients, when they are discharged the hospital.

"8. The patients, when received on the days of admission, should be placed in the wards which have been last ventilated and not in those that have been long inhabited ; where, it may reasonably be presumed, the air is considerably tainted.

"9. All incurable or infectious cases should be refused admittance ; and amongst these should be classed old chronic ulcers of the legs, and particularly those in which there is a great loss of substance, for these seldom remain long healed ; hence most hospitals are so crowded, that the intention of the charity is perverted, as the air is rendered unwholesome.

"10. All offensive, gangrenous, or other putrid sores, should be placed in distinct rooms provided for that purpose ; and not suffered to taint a whole ward.

"11. There should be particular rooms provided for those patients who are the subjects of operations ; they should be in the most airy situation, never long inhabited and alternately cleaned and ventilated, as before advised.

"12. A hospital should never be crowded on any account ; and always of so large a construction that some part of the building may at all times be uninhabited for the purpose of white-washing, ventilation, &c.

"13. When any person has been affected with a putrid disease, or confined to bed for a length of time, let the bed be emptied and the bed-stocks, the bed, the sheets, and other linen be washed, and the rest of the bedclothes exposed for some time in the open air and baked in the oven, before they be used again.

"14. Let the nurses see that every patient's hands and face are washed every morning ; and their feet, once a week.

"15. Let the nurse of each ward be liable to a fine, to be deducted from her wages, if some of the windows in her ward are not kept open during a stated number of hours every day.

"16. To every Infirmary, particularly where the wards are crowded, a house in the country, well situated, and at a convenient distance should appertain ; without such assistance many of the patients must perish, which would be easily and certainly preserved ; and it will be found the best policy in the trustees of an Infirmary

to provide such an appendix. By such assistance patients may be easily cured at a small expence ; but, if suffered to remain in an Infirmary, their recovery will



EDWARD ALANSON

either be prevented, or obtained in a great length of time at a considerable expence by the most costly drugs, nutritious diet, &c. Likewise the house will be constantly crowded with the most miserable objects, to the anxiety of those who attend them

and the exclusion of other patients, who might have been cured in the interim. Many hospital-surgeons are under the necessity of providing lodgings in the country, at their own expence, for their patients who have undergone operations; rather than suffer the pain of a disappointment in completing a good cure, or seeing their patient languish under a hectic, incurable in a crowded Infirmary. Therefore it is hoped that these considerations will influence the humane trustees, to provide these conveniencies for the poor sufferers."

It is evident that Mr. Edward Alanson, Surgeon to the Liverpool Infirmary in 1782, was almost exactly a century in advance of his time. There is no evidence that his suggestions were adopted at Liverpool or in any other hospital at home or abroad. There is evidence that he was a thoroughly good practical surgeon. He advocated amputation by a skin and muscle flap with circular amputation of the deep muscles when the majority of his contemporaries were content to remove a limb by the simple circular method. He tried to get union by first intention instead of by suppuration. He freed the arteries before he tied them when it was the custom to ligature nerves, arteries and the surrounding tissue in a single bunch, and he undertook with success such large operations as removal of the whole arm with the scapula. The first edition of his *Practical Observations* was a pamphlet of 64 pages, published in 1779; the second edition, enlarged to 296 pages, was published in 1782, and was translated into French by M. Lassus in 1784.

Alanson was born on Oct. 23, 1747, at Newton-le-Willows, Lancashire, the son of John and Margaret Alanson. His father was a large farmer and a man of substance. The son was apprenticed in 1763 to William Pickering, one of the Surgeons to the Liverpool Infirmary. He went to London in 1768, where he and Edward Jenner were amongst the first house pupils of John Hunter, who had just been elected Surgeon to St. George's Hospital. The fruits of his association with the Hunterian school are still to be seen in the Museum of the Liverpool University, where there is the body of a child with the label, "Injected and Dissected by Edw^d. Alanson in Docter W^m. Hunter's Rooms London March 1769."

Alanson returned to Liverpool in 1770, was elected Surgeon to the Infirmary, and in 1778 became one of the first three Surgeons at the Liverpool Dispensary. He lived with his former master, William Pickering, until in June, 1774, he married Miss Holland, daughter of a merchant. He joined practice with Henry Park (*vide* p. 205) in 1777, moved to Wavertree in 1790, resigned from the Infirmary in 1794 on the ground of ill health, retired to Aughton near Ormskirk, returned to Wavertree in 1808, and died there on Dec. 12, 1823, survived by his wife and five of his twelve children. It is noteworthy that neither he nor Henry Park ever became *Members of the Corporation of Surgeons*, though both styled themselves Surgeons.

I am indebted to Mr. R. W. Murray, F.R.C.S., late Honorary Surgeon to the David Lewis Northern Hospital and Liverpool Infirmary for Children, for information and for the portrait. He wrote in 1914 *Edward Alanson and his Times*.

ROTATION AT THE SHOULDER A CRITICAL INQUIRY

By LEE MCGREGOR

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[THIS paper was completed but for the drawings in April, 1936. By the last mail (June, 1936) I received Codman's¹ monumental work on the shoulder. To the best of my knowledge this is the first copy of the book to reach Johannesburg. I find that Codman has preceded me in the discovery of certain facts which I had thought described for the first time in this paper. This is not to be wondered at in so acute an observer as his work demonstrates this great American surgeon to be, and I gladly acknowledge the precedence I indicate above.

In his book Codman makes two statements to which in particular my remarks refer: (1) On p. 43—"you can prove that the completely elevated arm is in either extreme external rotation or in extreme internal rotation"; (2) On p. 44—"The range of rotation of the humerus diminishes as it is elevated".

As this knowledge is of practical value I submit my communication unchanged.]

SUMMARY OF CONTENTS

- I. INTRODUCTION
- II. REVIEW OF THE LITERATURE
- III. ROTATION OF THE HUMERUS DURING FLEXION
- IV. THE MID-POSITION AND THE ROTATION OF THE HUMERUS
- V. SURGICAL APPLICATIONS
- VI. OPTIMUM POSITION FOR TREATMENT OF ACUTE INJURIES
- VII. CONCLUSIONS
- VIII. SUMMARY

I. INTRODUCTION

The humero-scapular joint is distinguished from all other joints of the body by its extensive range of movement, by the complexity of these movements, and by the fact, unique in human anatomy, that it is overhung by an osseo-fibrous arch which exercises highly important limitations on certain of the excursions at the joint. So many factors require analysis in an understanding of the mechanics at the shoulder-joint that there are still aspects of the subject regarding which knowledge is incomplete. Furthermore, Ridlon² expressed the general opinion of surgeons anent shoulder injuries when he said "the difficulties encountered in the treatment of injuries to the shoulder-joint have always been more troublesome and more obscure than the difficulties in treating all other joints put together"; and Lovett³ remarked "that no common injury is so badly treated, save possibly the spine, as injuries to the shoulder". It is patent then, that any light on the manifold obscurities and perplexities of this inadequately understood joint may be of value in smoothing out some of these difficulties.

The objects aimed at in this paper are : (1) To indicate that our knowledge of rotation at the shoulder is incomplete ; (2) To amplify this knowledge ; (3) To make certain important surgical deductions from this fuller understanding.

II. REVIEW OF THE LITERATURE ON THE ROTARY PHASE OF SHOULDER-JOINT MOVEMENTS

Rotation of the head of the humerus receives but little space in the large modern text-books of anatomy, as the following extracts show.

*Text-book of pure anatomy*⁴ : "The humerus is rotated outwards by the infraspinatus and teres minor ; and it is rotated inwards by the subscapularis, latissimus dorsi, teres major, pectoralis major, and the anterior fibres of the deltoid ".

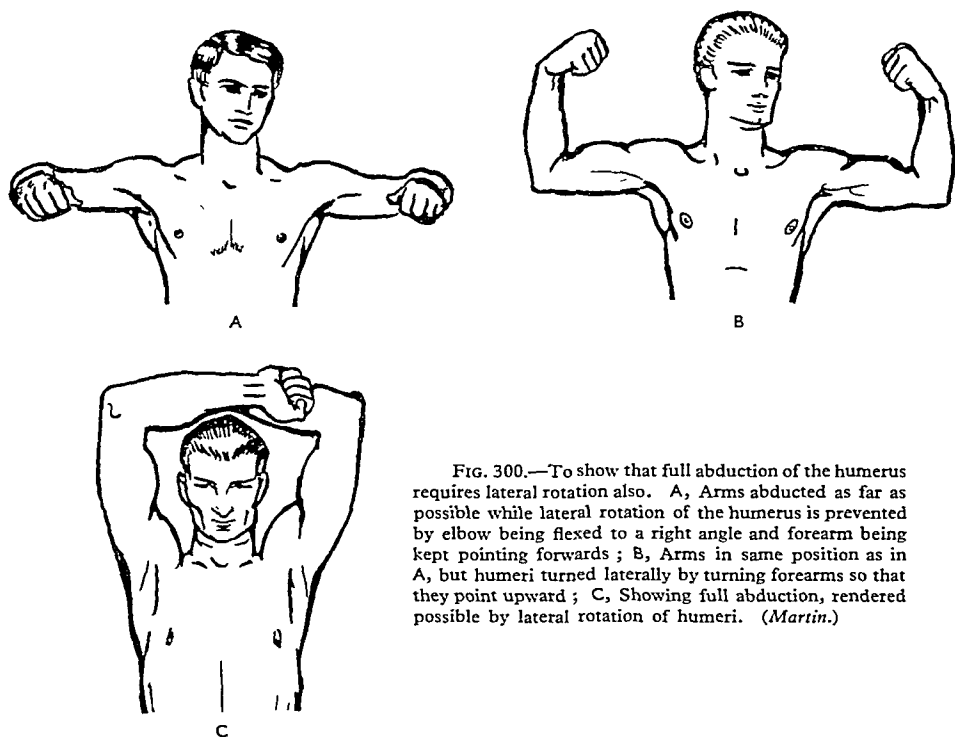


FIG. 300.—To show that full abduction of the humerus requires lateral rotation also. A, Arms abducted as far as possible while lateral rotation of the humerus is prevented by elbow being flexed to a right angle and forearm being kept pointing forwards ; B, Arms in same position as in A, but humeri turned laterally by turning forearms so that they point upward ; C, Showing full abduction, rendered possible by lateral rotation of humeri. (Martin.)

*Text-book of pure anatomy*⁵ : Rotation of the humerus takes place "round a vertical axis drawn through the extremities of the humerus from the centre of the head to the inner condyle ; in rotation forward (that is medial-ward) the head of the bone rolls back in the socket as the great tuberosity and shaft of the humerus are turned backward, i.e., lateral-ward". The muscles concerned in the actions are then indicated.

*Text-book of surgical anatomy*⁶ : "Rotation of the humeral head normally approximates 180 degrees ".

Text-book of orthopædic surgery: "Rotation of the shoulder is greatest when the arm is partially abducted, and in a dissected joint the arc may approximate 135 degrees. At a right angle with the body under these circumstances it is about 90 degrees."

All these excerpts are taken from text-books which because of their great general worth are used almost universally. It will be seen that rotation at the shoulder-joint is not extensively commented on.

In 1932 Martin,⁸ University anatomist at Trinity College, Dublin, made a contribution to the existing knowledge of rotation of the humerus which is quite the most important yet written. Surgeons taught for generations that most

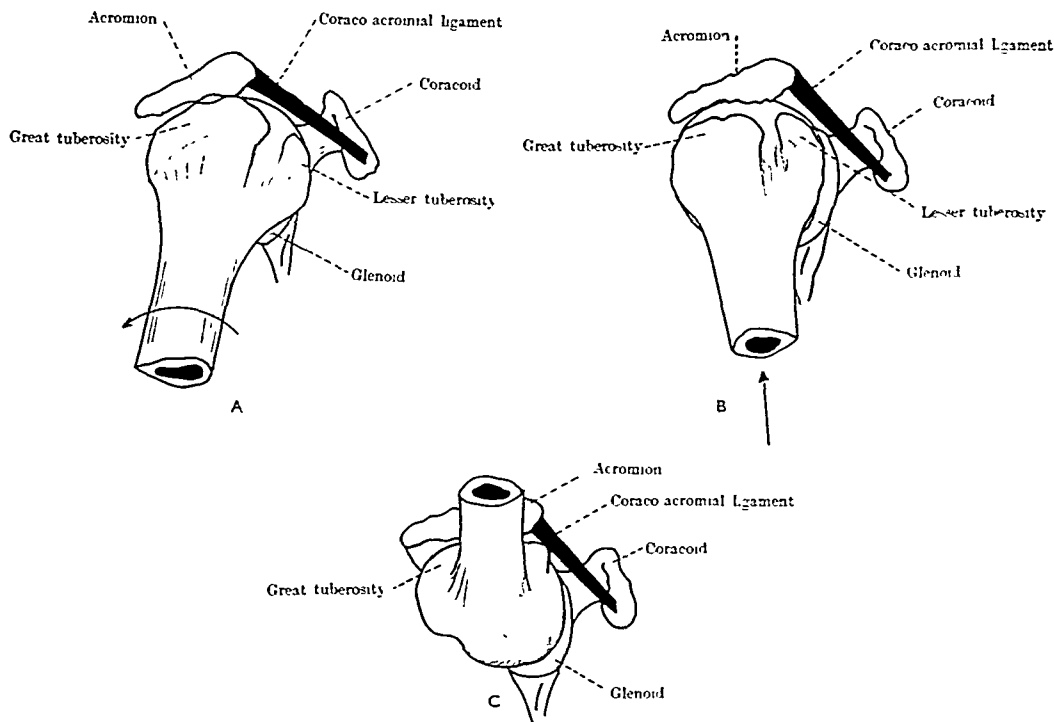


FIG. 301.—Demonstrating the part played by the osseo-fibrous arch overhanging the head of the humerus in the mechanism of abduction. A, The great tuberosity of the humerus is seen to impinge on the acromion; B, The humerus is rotated out; C, Abduction can now continue, as the tuberosity has receded beneath the acromion.

shoulder-joint injuries should be treated in abduction and full external rotation of the humerus, but it remained for an anatomist to show the scientific reason for this position of the bone.

The essence of Martin's paper is that, whereas abduction of the humerus to a right angle can be carried out whether the bone is rotated in or out, the second 90 degrees of abduction cannot be effected unless the humerus is fully rotated outwards. This is well shown in his lucid illustrations (*Fig. 300, A, B, C*). In *Fig. 300, A* the humerus is rotated inwards, being held in this position by the forearms being flexed to right angles and directed forwards. Very little further

abduction is possible. If the test is carried out on one side only, the result may be vitiated by tilting of the body. In *Fig. 300, B* the humeri are rotated out to the full, the forearms being flexed as before and directed vertically upwards. Now abduction can be carried to the vertical with ease (*Fig. 300, C*). Martin points out that during the second 90 degrees of abduction the great tuberosity of the humerus comes in contact with the lateral edge and under surface of the acromion and coraco-acromial ligament, and further abduction can only occur if the tuberosity slides under the acromion, which it can only do by passing backwards—i.e., lateral rotation of the humerus (*Fig. 301*).

The very important fact is adequately stressed that deficient abduction at the shoulder may be dependent on inability to effect lateral rotation of the humerus and not on any defect in the mechanism of abduction itself.

It has for a long time been held a wise precept that injuries of the shoulder-joint be treated in right-angled abduction and full external rotation of the humerus. That the explanation has followed many years after the principle of treatment was realized is due to the value of the latter having been appreciated, empirically, from the clinical observation of such cases. Surgical history can show many instances where empiricism has been justified later by scientific fact.

III. ROTATION OF THE HUMERUS DURING FLEXION

Carrying the upper limb straight forwards (in the sagittal plane of the body) up to the vertical is called flexion. Martin pointed out that whether the arm be carried to this position by flexion or by abduction (movement in the frontal plane), the position of the humerus at the end of the act is identical, i.e., the lateral condyle points straight backwards. The inference is that the mechanism of rotation of the humerus is the same in both instances. This is not the case. Execution of the following movements will readily demonstrate that whereas in abduction to the vertical the humerus rotates fully externally, *in flexion of the arm to the vertical, the bone rotates fully internally.*

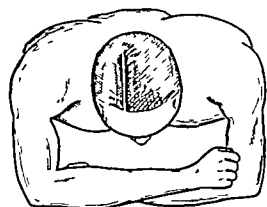


FIG. 302.—Arms and forearms horizontal, elbows flexed.

1a. Hold the arms vertically above the head, the elbows flexed to a right angle (*Fig. 300, C*); now bring them down to the horizontal position, i.e., right-angled abduction (*Fig. 300, B*). Note that in this position no further external rotation of the humerus can occur, indicating that at the vertical the humerus is fully rotated out.

1b. From the same position as in (1a) (vertical with elbows flexed), bring the arms down in the forward direction to the horizontal (*Fig. 302*). Note that in so doing no rotation of the humerus occurs, as indicated by the position of the lateral epicondyle being the same before and after the movement. Now rotate the forearms out (*Fig. 303*)—they readily go to the vertical—indicating that they have rotated outwards through a right angle, and that in the vertical position the humerus is fully rotated in.

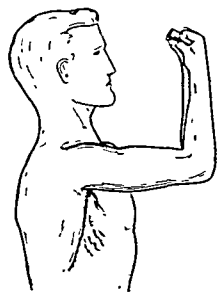


FIG. 303.—The same as *Fig. 302* but with the forearms rotated outwards to the vertical.

Here then is an apparent *reductio ad absurdum*. When the arm is vertical no rotation is possible in the shoulder-joint, yet at the end of abduction (dependent on full external rotation) the lateral epicondyle points straight back. The explanation is simple, and is due to the fact that the one movement—i.e., flexion—is carried out in a plane a quarter of the circumference of a circle (90 degrees) in front of the plane of the other movement, abduction.

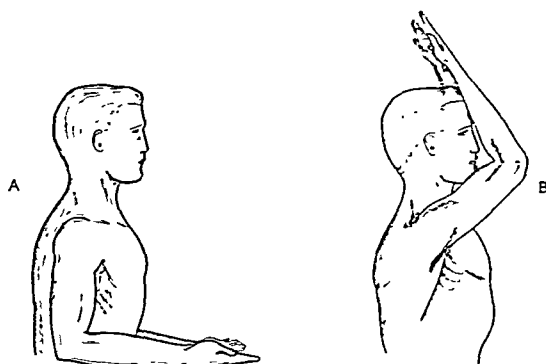


FIG. 304.—Showing that with the humerus rotated out the arms cannot be flexed to the vertical.

2a. Hold the elbows against the body, the forearms bent to a right angle on the arms and pointing forwards (Fig. 304, A). Now flex the arms by carrying them straight up in a forward direction. They can be carried up only to a right angle and a half because the humeri are rotated out (Fig. 304, B). The arms can be forced further up, but only by increasing the lumbar lordosis and tilting the shoulders.

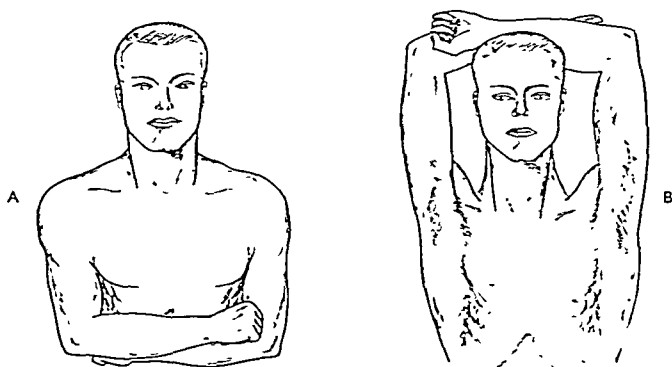


FIG. 305.—Showing that with the humerus rotated in, flexion to the vertical is possible.

2b. Holding the arms to the side, the elbows flexed to a right angle, and the forearms lying against the abdomen (Fig. 305, A), now flex the arms: they pass without effort to the vertical position (Fig. 305, B), because the humeri are fully rotated in.

Explanation.—The reason why internal rotation is essential to full flexion is as follows: when the arm is flexed in external rotation, the lesser tuberosity

of the humerus covered by the subscapularis impinges against the costocoracoid ligament and can only roll under this obstruction by rotating in, and then flexion can be completed (*Fig. 306*).

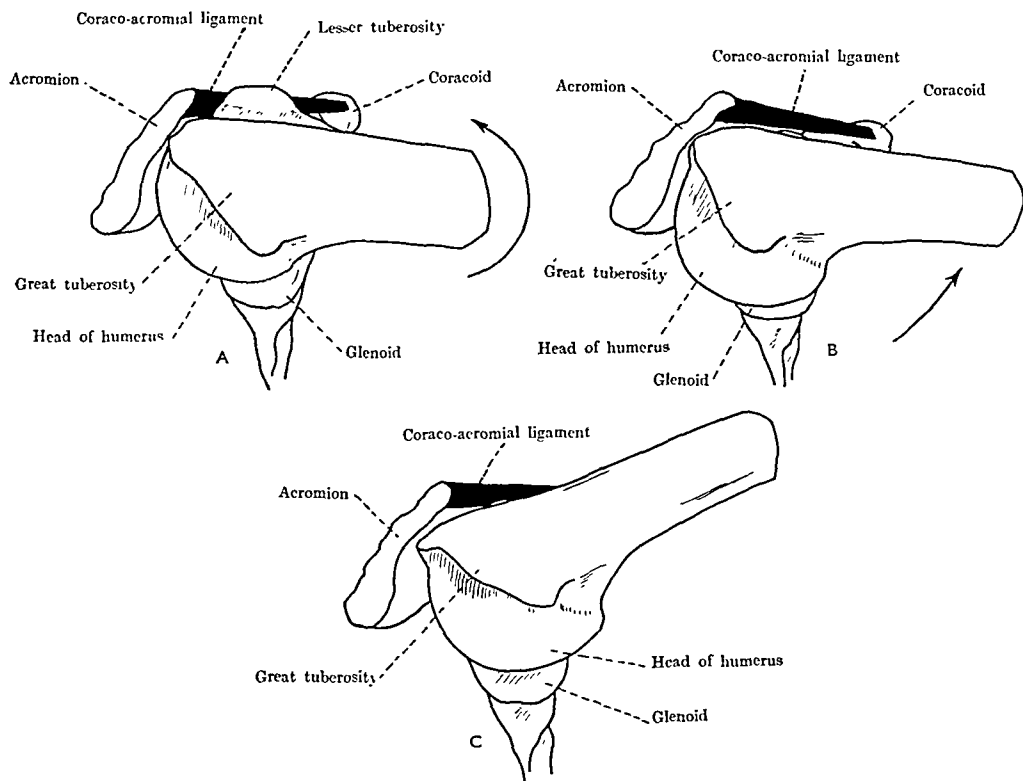


FIG. 306.—Showing that in flexion of the humerus to the vertical the lesser tuberosity impinges against the coraco-acromial arch, and the movement can only be completed when the tuberosity glides under the arch by rotating the humerus medially or inwards. A, In right-angled flexion the lesser tuberosity is obstructed by the coraco-acromial ligament when the arm is rotated out; B, With the arm rotated in, the tuberosity slips beneath the ligament; C, Further flexion is now possible.

The fact being established that flexion of the arm is as dependent on internal rotation as abduction is on external rotation, certain other important deductions can be made: (1) As to the mid-position of the humerus; and (2) As to the rotation of the humerus in different positions of the upper limb.

IV. THE MID-POSITION AND THE ROTATION OF THE HUMERUS

The Mid-position.—Holding the arm in 90 degrees abduction (horizontal in the frontal plane), the elbow bent to a right angle, the forearm directed forwards, observe that very little further abduction is possible (*Fig. 307, A*). Now bring the whole upper limb forward a few degrees—note that the limb can be elevated higher (*Fig. 307, B*). As the limb swings forward it can still be further elevated, until finally with the arm pointing straight forward and the elbow held at right

angles in front of and parallel to the transverse diameter of the thorax, the arm can be elevated to the vertical (full flexion) (*Fig. 307, C*).

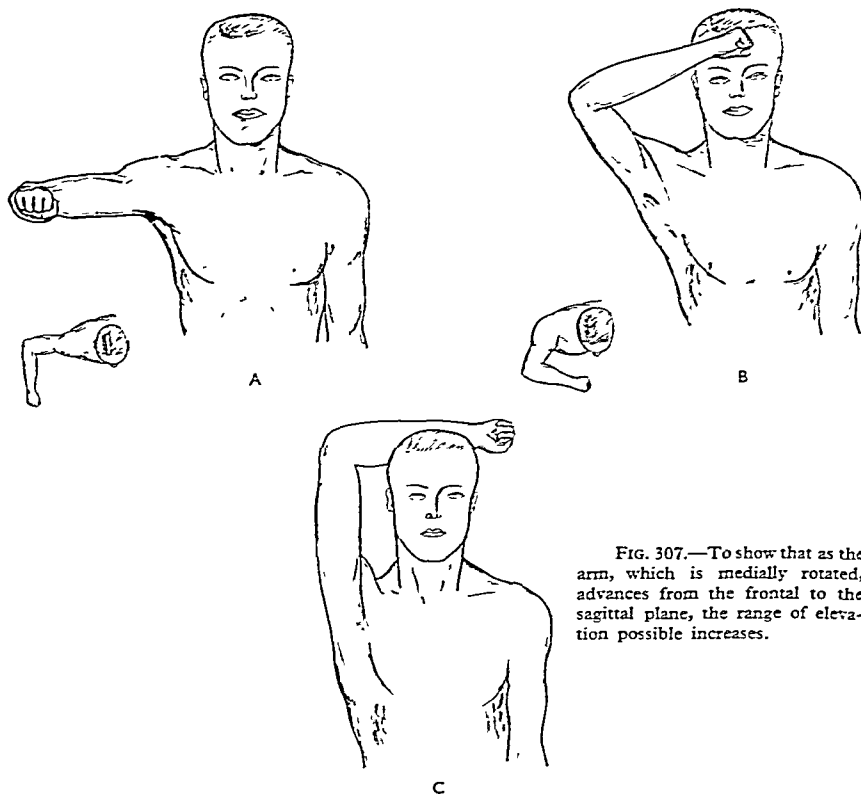
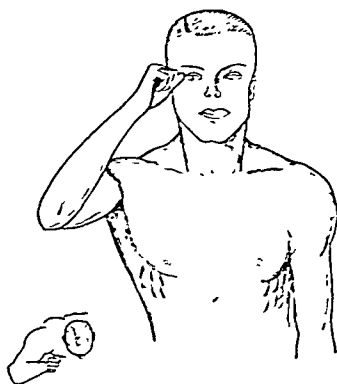


FIG. 307.—To show that as the arm, which is medially rotated, advances from the frontal to the sagittal plane, the range of elevation possible increases.

In the mid-position between right-angled abduction and right-angled flexion, the humerus can be elevated to a right angle and a half, and this range remains

FIG. 308.—The mid-position of the humerus. This is the optimum position for the treatment of most lesions at the shoulder-joint.



the same whether the bone be rotated in or out. This is therefore the mid or neutral position, and it may be obtained with the humerus in any position of rotation (*Fig. 308*).

Rotation of the Humerus in Different Positions of the Upper Limb.—

- a.* When the arm is vertical no rotation of the humerus is possible (*Fig. 309, A*).
b. When the arm is at right-angled abduction or flexion, rotation of the humerus can take place through a range of just over a right angle (*Fig. 309, B*).
c. With the arm hanging at the side rotation is possible through a range of one and a half right angles (*Fig. 309, C, D*). In no position can rotation through two right angles be obtained at the shoulder.

It is then established that with the arm at the vertical no rotation is possible, but as soon as the limb begins to move downwards (whether in the frontal or sagittal plane) rotation becomes possible, and its range increases until the maximum is attained with the limb dependent.

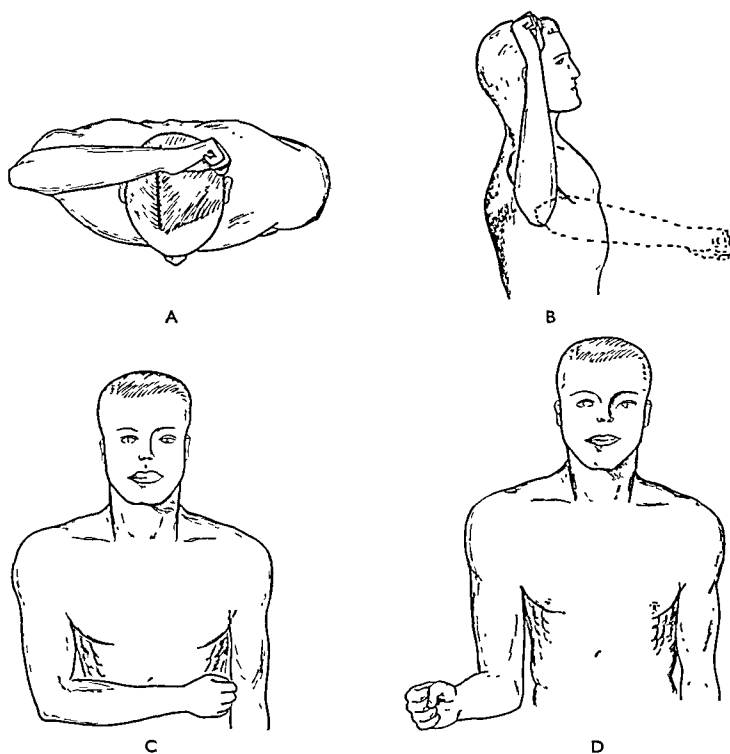


FIG. 309.—A, With the arm in full (vertical) abduction, no rotation of the humerus is possible—i.e., the joint is locked in so far as rotation is concerned; B, With the arm at a right angle, the range of rotation is shown by the interrupted line; C and D show the range of rotation with the arm at the side.

Explanation.—As pointed out above, in full abduction the humerus rotates out, the great tuberosity rotating under the acromion; so too in full flexion the small tuberosity rotates under the coracoid process. Each of these final positions is therefore a 'locking-home movement', and rotation is impossible until the joint is unlocked. Once the limb commences to move downward rotation begins at once. It would appear that this fact is an unanswerable argument in support of the contention of Cathcart⁹ and Lockhart¹⁰ that the humerus moves on the scapula throughout the full range of abduction and flexion and not only in the first 90 degrees of these movements.

In considering the movements of flexion and abduction at the shoulder in our daily activities it is very soon evident that a position which is very seldom made use of is that of right-angled abduction in the frontal plane combined with external rotation, or any degree of abduction beyond a right angle. The position may be used by some in serving at tennis or in stretching when sleepy. In doing her back hair, a woman rotates her arm out, but the position of the humerus is nearer the mid or neutral one than that of right-angled abduction (*Fig. 310*). In reaching for something on a shelf—e.g., a book—the arm goes up in the neutral position. Even in bringing the hand to the mouth the humerus is in a plane mid-way between flexion and abduction (*Fig. 311*).

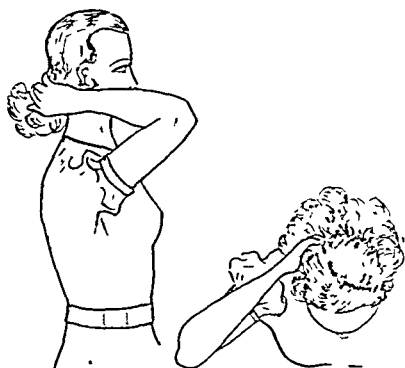


FIG. 310.—To show that in doing her back hair a lady's arm is in the mid-position.



FIG. 311.—Position of the arm in carrying the hand to the mouth in eating.

A consideration of the actions performed daily will show that human beings seldom use the movements of pure flexion or abduction, but move the humerus in some plane between these two extremes, the most generally useful being a plane about mid-way between the two. This section may be concluded with the statement that the movement of abduction in the frontal plane plus full external rotation of the arm is an action which is seldom required and which could be dispensed with with little resultant loss to the efficiency of the limb, providing that elevation of the arm could be performed in a plane anterior to that of the transverse axis of the body. If the arms are held in right-angled abduction and full external rotation for a few moments it is readily appreciated that the posture is forced and uncomfortable.

V. SURGICAL APPLICATIONS

The diseased or injured shoulder if neglected takes up a position of adduction and internal rotation. Such a limb is of little use to its owner. To prevent this disability occurring it is everywhere recognized that in the treatment of such cases the arm must be abducted from the side. Some authors advise that all injuries of the joint (reduced dislocations excepted) should be treated in abduction and external rotation. In fact the position of right-angled abduction with full external rotation receives the widest acceptance as the optimum position for the treatment of lesions of the shoulder-joint which are not considered likely to end in ankylosis.

Jones and Lovett,¹¹ discussing the treatment of sprains of the shoulder, state : " In the case of the shoulder, the grave after-effects are due to limitation of abduction and internal or external rotation. For this reason, and because the removal of the weight of the arm expedites recovery, and because the possibility of adhesions is minimized, the arm should be treated in the abducted position, and preferably externally rotated by means of an abduction or platform splint." Quotations giving the same advice may be made from many text-books. The medical student will answer without hesitation that shoulder-joint injuries must be treated in right-angled abduction and full external rotation. A method of treatment which receives such powerful and general support as that quoted must have strong arguments in its favour. Effort will be made to show that the advice is nearly correct, but that it may be improved in some degree with vast increase in the comfort of the patient.

The Disadvantages of the Position of Right-angled Abduction with Full External Rotation.—Because of the peculiar anatomical features of this joint, particularly the osseo-fibrous arch which overhangs it, and the large important tendons and muscles which lie on or are incorporated with the joint capsule, it is readily appreciated that there is only just room normally for the execution of the complex movements of the humeral head beneath the overhanging arch. When the joint is sprained, for example, as with the tearing of muscle or tendinous fibres, there is an infiltration in and around the joint capsule, so that movement at the joint causes pressure by the over-hanging arch on exquisitely sensitive structures. This pain increases as the pressure increases. Pressure is greatest as tendons attached to the tuberosities pass under the coraco-acromial arch in the position of right-angled abduction. External rotation introduces the added factor of tension on the ligaments on the front of the joint and the medial rotators such as the subscapularis. Were a deliberate effort made to choose the position of greatest discomfort, it is probable that the position of right-angled abduction in the frontal plane with full external rotation would be the position.

Is this position more uncomfortable than may be accounted for by the combination of injury plus fixation of a joint which has hitherto been normal? In answer to this question : Two doctors practising in Johannesburg sustained injury to the shoulder and were ' put up ' in right-angled abduction. Each stated that the pain was intolerable, and that not even the fear of subsequent disability could induce them to continue wearing the splints. In private and hospital practice it is similarly found that many patients will not tolerate this position after acute joint injuries. Sir Robert Jones,¹² speaking of splints to maintain abduction at the shoulder, says : " Of these none has proved satisfactory, for no matter how comfortable the splint may seem when the surgeon tries it on himself, it is a very different thing when put on a patient with a painful shoulder-joint ". The fact that some, particularly hospital, patients will wear aeroplane splints for weeks is a remarkable example of human fortitude. It is probable that apart from the rigid, unalterable fixation of the joint, it is the forced unnatural position entailing pressure and tension which causes such torture to the patient.

After fractures of the surgical neck of the humerus, the upper limb is often put in extension on a Thomas arm splint in right-angled abduction and external rotation. Such a patient is nothing like so uncomfortable as the one who wears an aeroplane splint, and this for several reasons. The extension in itself probably

acts as a distraction force to those factors making for tension under the coraco-acromial arch, and, furthermore, the patient can move his body in relation to the splint, at the shoulder, and thus avoid rigid immobilization; moreover, the injury is distal to the joint capsule, and the damage done so considerable that extravasated blood may track for some distance and relieve pressure. In cases of paralysis around the shoulder an aeroplane splint is readily tolerated. Here the matter is entirely different. Owing to paralysis the head of the humerus may fall away from the coraco-acromial arch, perhaps not very much, but enough to avoid any element of tension arising.

It has been shown that right-angled abduction in the frontal plane with full external rotation is a position almost never used naturally, that it is a forced position,

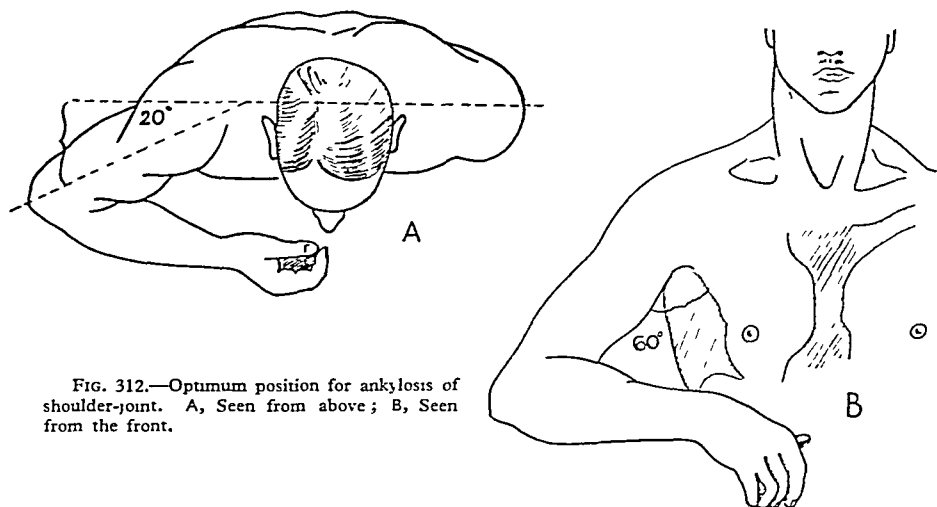


FIG. 312.—Optimum position for ankylosis of shoulder-joint. A, Seen from above; B, Seen from the front.

and that its loss would result in little if any disability. If, therefore, a position can be found which is more comfortable and which is sound from the view-point of subsequent function, that will be the optimum position for the treatment of acute shoulder injuries.

It must be made clear that this communication is not concerned with the optimum position for *ankylosis* at the shoulder, but with that position which will combine minimal discomfort with maximal subsequent function. The optimum position for ankylosis is 45 to 90 degrees abduction and 20 degrees flexion in front of the coronal plane (*Fig. 312*). The younger the patient the greater the abduction.

VI. THE OPTIMUM POSITION FOR TREATMENT OF ACUTE INJURIES OF THE SHOULDER-JOINT

The desiderata in arriving at the best position for the treatment of such injuries are:—

1. The anti-gravity muscles—i.e., abductors—must be put at rest. These are the supraspinatus and deltoid.

2. Should a muscle be torn, the position chosen must secure relaxation of the injured muscle and consequent approximation of the torn ends. The muscles

most often torn at the shoulder are the supraspinatus tendon and that of the long head of the biceps.

3. The position must avoid adhesions in the lower dependent pouch of the joint capsule.

4. The position must be such that if stiffness occurs it will be as little disabling as possible; also it must be one which will be advantageous to start from in securing a wider range of movement.

It is submitted that on anatomical, physiological, and functional grounds this optimum position is that shown in *Fig. 308*.

Advantages of the mid-position.—

1. The arm is at right angles to the body mid-way between the position of right-angled abduction and right-angled flexion.

2. The forearm is in the mid-position between full external and full internal rotation.

In this position the supraspinatus is relaxed, so also is the biceps. Neither of the tuberosities of the humerus is engaged beneath the coraco-acromial arch; pressure is therefore avoided. Also the capsule and its ligaments and the rotators of the joint are relaxed; tension is therefore avoided.

It will often be found, particularly in the condition of 'peri-arthritis' or bleeding round the joint, and sprain of the supraspinatus, that the patient has difficulty and pain in performing elevation of the arm in the frontal plane, but can readily elevate in a plane anterior to this. This is of obvious importance in treatment.

The succession of little injuries inflicted by efforts at performing abduction in the frontal plane should be avoided. The harmful effects of such repeated minor traumata have been splendidly elaborated in Mr. Watson Jones's¹³ recent brilliant exposition. The mid-position can be secured by a splint fixed to the trunk, or by plaster-of-Paris, the latter being more efficient than the former.

It is interesting to see how this position, arrived at by the study of the anatomy and physiology of the joint, accords with the opinions of some observers who have not been satisfied with the routine use of abduction in the frontal plane plus full external rotation. Dickson and Crosby,¹⁴ discussing the treatment of peri-arthritis of the shoulder-joint, say that they find the position of full abduction has no particular advantage and is likely to add to the patient's discomfort (their figure, however, shows full right-angled abduction). They advise traction in moderate abduction and external rotation. In the discussion which ensued on the presentation of this paper abduction and external rotation were generally advised in the treatment of peri-arthritis. Brockman,¹⁵ speaking of the abduction splint, says that it is at the best an uncomfortable instrument and requires continuous adjustment. Bankart,¹⁶ in his book on manipulative surgery, states that, after the manipulation of the shoulder-joint in which adhesions have been formed, "there is no conceivable object in making the patient 'come round' from the anæsthetic with the arm in a position of abduction and external rotation". Nowhere in his book can I find that this position is advised. Böhler¹⁷ strongly advises that severe sprains and contusions of the shoulder be treated with the joint in the mid-position—that is, "90 degrees abduction, 90 degrees outward rotation, and 30 to 40 degrees in front of the frontal plane". In this position, he states, all the muscles of the shoulder-joint lie in mid-position, pain disappears at once, and active movements are again possible (*Figs. 313, 314*).

Speaking generally, right-angled abduction, with varying degrees of rotation, has been and is advocated for certain nerve lesions, severe sprains and contusions of the shoulder, certain tendon ruptures, and some fractures round the joint. The mid-position may with advantage replace right-angled abduction in the frontal plane in all these lesions, for the reason that it is much less irksome and that it secures better muscle relaxation. Böhler¹⁸ strongly advises the mid-position for fractures of the surgical neck of the humerus with displacement, as in this position the pectoralis major muscle is relaxed and does not tend to pull the reduced fragments into faulty position.



FIG. 313.—Position advocated by Böhler for the treatment of severe sprains of the shoulder and of fracture of the surgical neck of the humerus. (After Böhler.)

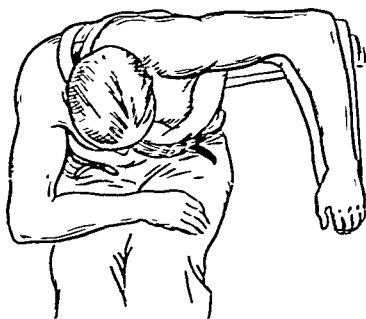


FIG. 314.—Figure showing faulty position for the treatment of fracture of the surgical neck of the humerus. (After Böhler.)

When, however, traction is necessary in the treatment of fractures round the shoulder, it may be more convenient to apply extension on a Thomas abduction splint in the frontal plane. For reasons which have been given above this is a much less irksome form of splintage than that obtained by an aeroplane splint. It would be difficult to apply heavy traction to the humerus in the mid-position, as the extension would be awkward to secure in this plane and the counter-extension equally difficult. So, too, with some cases of fracture of the great tuberosity of the humerus with displacement, reduction might be better secured by the use of a Thomas abduction splint.

VII. CONCLUSIONS

The distinctive and unique features of the shoulder-joint are :—

1. The fact that it is overhung by a closely applied osseo-fibrous arch.
2. The architecture of the joint is such that there is only just room for joint movements beneath this arch. Any peri-articular swelling, such as follows a sprain or contusion, means that tension and pressure occur in those movements during which the humeral tuberosities slide beneath the coraco-acromial arch, the result being pain in the execution of such movements.
3. Rotation of the humerus is an essential part of the movements of abduction and flexion above the horizontal plane ; so much so that abduction above 90 degrees cannot be executed unless the humerus is fully rotated out, and the last 50 degrees of flexion are equally dependent on internal rotation.

These features distinguish the mechanics of the shoulder-joint from all other joints ; true, lateral flexion of the spine cannot occur without rotation : however,

not one, but many joints are concerned in this action; rotation of the femur when the psoas flexes the hip-joint is the nearest analogy in the body, but here the joint architecture is of a much simpler type than at the shoulder. Rotation being essential to the free movements at the joint, the correct appreciation of this phase of joint activity becomes of paramount importance in the superintendence of cases of disease or injury at the shoulder. As the range of rotation of the humerus is at its maximum with the arm at the side of the body, it is reasonable to instruct the physio-therapist to exercise the rotary function of the joint with the limb in this position, more especially as the sufferer will tolerate this movement better here than with the limb elevated. Abduction in the frontal plane, particularly if combined with full outward rotation of the arm, is an extremely uncomfortable position for a recently injured shoulder; moreover, it is a position which is of hardly any economic value, and should therefore be discontinued as a method of rigid fixation. In cases of peri-arthritis, the mid-position is that which is indicated on anatomical and physiological grounds, and is, furthermore, a position which can be assumed or maintained with far less discomfort.

VIII. SUMMARY

1. Full outward rotation of the humerus is essential to the performance of full abduction (Martin).
2. Full inward rotation of the humerus is essential to the performance of full flexion.
3. With the arm vertical no rotation of the humerus is possible. The degree of rotation increases from above down and is maximal with the arm at the side.
4. Horizontal abduction in the frontal plane combined with full external rotation of the arm is a movement seldom if ever used.
5. The position defined in (4) is one which causes extreme discomfort.
6. Fixing the humerus in this attitude in injuries at the shoulder should therefore be discontinued.
7. The mid-position is that in which the joint should be splinted. It is anatomically and physiologically sound and much less irksome.

REFERENCES

- ¹ CODMAN, E. A., *The Shoulder*, 1934. Boston, U.S.A.
- ² RIDLON, JOHN, *Surg. Gynecol. and Obst.*, 1932, 54.
- ³ LOVETT, R. W., *Ibid.*, 1922, 34.
- ⁴ GRAY, H., *Anatomy*, 1918, 371. London.
- ⁵ MORRIS, *Human Anatomy*, 1915, 257. London.
- ⁶ CALLANDER, *Surgical Anatomy*, 1933, 793. London.
- ⁷ JONES and LOVETT, *Orthopædic Surgery*, 1929, 54. London.
- ⁸ MARTIN, C. P., *Brit. Jour. Surg.*, 1932, xx, 61.
- ⁹ CATHCART, *Jour. Anat. and Physiol.*, 1884, xviii.
- ¹⁰ LOCKHART, R. D., *Jour. Anat.*, 1930, xiv, 288.
- ¹¹ JONES and LOVETT, *Loc. cit.*, 55.
- ¹² JONES, SIR ROBERT, *Orthopædic Surgery of Injuries*, 1921, ii, 437. London.
- ¹³ JONES, WATSON, *Brit. Med. Jour.*, 1936, i, 925.
- ¹⁴ DICKSON, J. A., and CROSBY, E. H., *Jour. Amer. Med. Assoc.*, 1932, Dec. 31, 2252.
- ¹⁵ BROCKMAN, E. P., *Practitioner*, 1932, 101.
- ¹⁶ BANKART, A. S. BLUNDELL, *Manipulative Surgery*, 1932, 131. London.
- ¹⁷ BÖHLER, L., *The Treatment of Fractures*, 1935, 179. Bristol: John Wright & Sons.
- ¹⁸ BÖHLER, L., *Ibid.*, 184.

MALLET FINGER

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It is the purpose of this paper to describe a rational method of treatment of the mallet or drop finger, and, at the same time, record the conclusions which have resulted from the experience of treatment in a series of 35 consecutive cases.

ETIOLOGY

Rupture of the extensor tendon at its insertion into the base of the terminal phalanx is the commonest of the subcutaneous tendon ruptures, and it was noted in the series under review that the injury was encountered once in 900 cases at a large Surgical Out-patient Department. The rupture is produced by indirect injury, the actively contracting tendon being subjected to a sudden passive flexing force, or less commonly to direct violence, the tendon being nipped against the dorsal aspect of the base of the phalanx. It is thus common in cricket, football, and baseball players; but such an apparently trivial accident as might occur during the making of a bed is frequently recorded as the cause of the injury. The history of having heard or felt the tendon snap and having experienced a sharp pain in the region of the distal interphalangeal joint is frequently given, but if the hand has been cold, the condition of the finger may not be noticed until some time afterwards.

SYMPTOMS

The injury is often diagnosed as a stave, occasionally as a dislocation, and strapped or splinted accordingly, and it is only on the removal of the dressing that it is realized that dorsiflexion is no longer possible. As seen shortly after the accident, there is swelling and tenderness especially on the dorsal aspect of the interphalangeal joint, the swelling tending to mask the flexion of the terminal phalanx which is present. Complaint is made of a dull, aching pain which is transformed into a sharp stab when the injured tendon is inadvertently contracted along with the uninjured extensors. Active extension in the complete lesion is impossible.

CLINICAL TYPES

Four separate clinical types can be recognized :—

Type 1.—Incomplete loss of extension due to a partial tear or stretching of the expansion. There is no demonstrable radiological lesion.

Type 2.—Absolute loss of extension, due to a complete tear in the expansion, but with no demonstrable radiological lesion (*Fig. 315*).

Type 3.—Absolute loss of extension with a demonstrable radiological lesion consisting of a chip of bone, often triangular in shape, separated from the base of the phalanx (*Fig. 316*).

Type 4.—A type seen in children—often the result of extreme violence—consisting of separation of the epiphysis at the base of the phalanx (*Fig. 317*). This type may be compound.

TREATMENT

A vast number of methods of treatment are in existence, most of which attempt by means of metal, wood, or celluloid splints to keep the terminal phalanx in extension. They have as obvious disadvantages imperfect immobility, clumsiness, tendency to pain from pressure, ease of removal by the patient on the slightest



FIG. 315.—Type 2. Showing flexion of the terminal phalanx but with no demonstrable radiological lesion.



FIG. 316.—Type 3. Showing the chip of bone separated from the base of the terminal phalanx.

discomfort, complete disability of the hand during the whole period of treatment, and the fact that the position is anatomically unsound from the point of view of healing, the central slip of the extensor being liable to tension when the sound fingers extend.

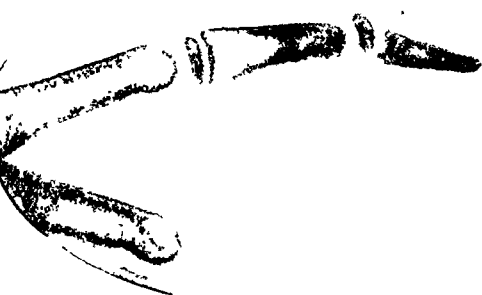


FIG. 317.—Type 4. Showing separation of the epiphysis.



FIG. 318.—Type 4. In plaster after reduction.

The most reasonable position for healing must be right-angled flexion at the proximal interphalangeal joint with hyperextension at the terminal interphalangeal joint, for in this position active extension is impossible, the central slip of the tendon being relaxed.

To obtain this position, securing at the same time absolute immobility with the minimum loss of function in the hand during treatment, a plaster case is ideal. The method eventually evolved for the application of the plaster case is as follows :—

A piece of Cellona plaster bandage about two feet long is rolled into a tube of such inside diameter as to roughly fit the finger. The tube should be rather longer than the finger to be treated, and should be cut on the slant at one end, so that it may fit closely at the web (*Fig. 319*).

The patient is shown how to press the thumb against the palmar surface of the terminal phalanx so that the proximal interphalangeal joint is a right angle and

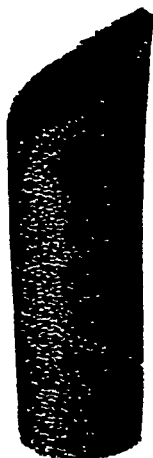


FIG. 319.—The plaster tube.



FIG. 320.—The position in which the patient is instructed to hold the finger.

the terminal phalanx hyperextended. A few moments spent on instruction are not wasted (*Fig. 320*).

The finger is now inserted into the dry tube of plaster (*Fig. 321*) and the hand dipped momentarily in warm water. The finger with its wet plaster covering is then squeezed tightly in the hand of the surgeon, and the patient is instructed to take up the required position until the plaster dries (*Fig. 322*).

If this technique is followed accurately, the operator especially refraining from holding the finger in position himself, no trouble will result from pressure, and in this series no complications have arisen other than the breaking of a plaster from a fall on ice and the return of four patients to have the splint replaced, being under the erroneous impression that plaster-of-Paris is waterproof. Many report their ability to carry on their normal occupations, even to the driving of a heavy motor lorry.

At the end of the period of immobility, which should be not less than five weeks, the case is removed by soaking in water to facilitate the straightening out of the right-angled bend, after which withdrawal of the finger is easy. Some cases show immediate full dorsiflexion, while others, although showing active extension against resistance, are still some degrees short of the normal. No estimate of the end-result should be made until about three weeks have elapsed, when with use many cases will be found to have regained the normal range of movement. No trouble has been experienced with persistent stiffness at the proximal interphalangeal joint, and it is probable that the rapid mobilization of the joints in the hands of



FIG. 321.—The dry plaster tube applied to the finger.



FIG. 322.—The finger held in the required position until the plaster dries

a masseuse is inadvisable, as the scar tissue is still stretchable, and full passive flexion of the terminal phalanx predisposes to a poor result.

PROGNOSIS

Prognosis depends on: (1) The time which has elapsed since the injury; (2) The age of the patient; (3) The type of injury.

With regard to the type of injury, *Type 1* gives excellent results without deformity. *Types 3* and *4* can be relied upon to give good results, but with some residual thickening at the terminal interphalangeal joint. The results in *Type 2* vary, so that the prognosis must be more guarded, for although the joint is of necessity opened into in *Types 2* and *3*, in *Type 2* there is always the possibility

DETAILS OF 35 CASES OF MALLEY FINGER

INITIALS	AGE	SEX	FINGER	DAYS SINCE INJURY	X RAYS	TYPE	RESULT OF TREATMENT
I. S.	26	M	R 3	5	Pos.	3	Full extension
J. McL.	13	F	R 3	11	None	2 or 3	Extension slightly less than other side
Mrs. L.	49	F	R 3	1	None	2 or 3	Very few degrees of full extension
I. S.	22	F	L 4	5	Pos.	3	Full extension
J. B.	13	M	L 4	4	Neg.	2	Few degrees short of full extension
Mrs. M	40	F	R 3	35	Pos.	3	Active extension but not full
P. S.	24	M	R 3	150	Neg.	2	Put up in plaster as experiment; no indication for operation; definite active extension obtained
Mrs. P.	33	F	L 4	7	None	2 or 3	Extension almost full
S. P.	21	M	L 2	2	None	2 or 3	Full extension
L. A.	29	M	L 3	14	Neg.	2	Strong active, but incomplete, extension
J. G.	50	M	R 4	9	Pos.	3	End-result unknown
Mrs. B.	34	F	R 3	28	Neg.	2	Operation: Good result, but extension a little short of full
Dr. B.	36	M	R 4	5	Neg.	2	Complete extension
J. A.	12	F	L 4	2	Neg.	2	Complete extension
Mrs. S.	65	F	R 3	?	Neg.	3	Extension almost full
B. O.	14	F	R 3	24	Neg.	2	A few degrees short of full extension
Mrs. M.	35	F	R 4	21	Neg.	2	Full extension
J. F.	24	M	R 3	21	None	2 or 3	End-result unknown
J. B.	31	M	L 2	7	Neg.	1	End-result unknown
A. M.	20	M	L 5	1	Pos.	3	Full extension
R. A.	42	M	R 5	7	Neg.	2	End-result unknown
W. McL.	21	M	L 4	28	Pos.	3	Full extension
C. B.	?	F	R 2	28	Neg.	2	Some improvement in function
R. B.	36	M	R 5	36	Neg.	2	Strong active extension, but not quite full
F. T.	41	M	R 5	28	Neg.	2	Patient did not wish treatment
H. T.	23	M	L 5	28	Neg.	1	Full extension
N. L.	35	M	L 5	12	Neg.	2	Operation: Following this, patient removed plaster and dressing; joint became infected; end-result—ankylosis in semiflexed position
T. H.	12	M	R 2	3	Pos.	4	Compound injury—operation—perfect result
D. L.	12	M	R 4	1	Pos.	4	Perfect result
Miss T.	47	F	R 5	21	Neg.	2	Strong active extension, but not quite full
M. L.	13	M	R 5	28	None	2	Operation: Full extension
R. G.	38	M	R 2	2	Neg.	2	Strong active extension, but not full
M. C.	61	F	R 2	2	Pos.	3	Not quite full extension
J. S.	15	M	R 4	12	Pos.	4	Perfect result
(J. D.	18	M	R 2	35	Pos.	3	Conservative treatment; some active extension obtained
(J. D.	18	M	R 3	35	Pos.	3	Operation: Almost but not quite full extension—very much better than finger treated conservatively

that the torn end of the proximal portion of the tendon has turned into the joint through the rent in the capsule, predisposing to disappointing results.

OPERATIVE TREATMENT

Lack of uniformity in the results obtained with conservative measures in the complete lesion without fracture makes operative measures worthy of consideration ; but as healing with some degree of dorsiflexion can always be obtained by conservative treatment, it is evident that ample justification for operation is only present where occupation or æsthetic considerations demand that a chance of a perfect result be offered.

Old Cases.—For the reasons outlined above, many patients do not report till some weeks after the injury, and it will be seen from the Table that a considerable proportion of cases fall into this category.

During the five weeks following the injury, some improvement in function and deformity may be expected by immobilization in plaster as in the fresh case, so that indications for operation are essentially as for the fresh case falling into *Type 2*.

Indications for Operative Treatment.—It follows from the above that operation should be reserved for :—

1. Compound injuries—often epiphysial separations, and referred to here as *Type 4*.
2. Certain fresh cases belonging to *Type 2* where at least a chance of full dorsiflexion is essential.
3. Certain old cases which demand a chance of increased dorsiflexion.

The Operation.—The success of the operation depends on the use of a plaster case applied in exactly the same manner as in the fresh injury ; hence the necessity for instructing the patient in the method of application and retention and the use of local infiltration anæsthesia at the base of the finger.

The compound injury is treated by the usual methods. Where the diaphysis has been torn away from the epiphysis the base of the nail has probably been evulsed and will require removal. No sutures are inserted in the capsule or extensor expansion, apposition of the torn edges being maintained by hyperextension alone.

The best incision for the deliberate operation has been found to be L-shaped, the short limb crossing the dorsum of the finger transversely, just proximal to the base of the nail, the long limb being dorsi-lateral, extending in a proximal direction (*Fig. 323*). This incision gives good exposure, while the sloughing which often follows the use of the U-shaped incision, and the disfiguring and sensitive scar which follows the midline dorsal incision, are avoided.

While the skin-flap is being dissected up, care must be taken not to damage the capsule or the remnants of the torn expansion. Procedure now will depend on the type of case. In the fresh case where the proximal end of the torn expansion has turned into the joint, the edges should be approximated, and two sutures of 0000 catgut mounted on the smallest usable curved needle inserted. If suture is impossible, as it often is, the edges should merely be approximated and the terminal phalanx maintained in hyperextension.

In the old case a previous chip fracture or tear may be reproduced and the edges freshened, suturing where possible, otherwise maintaining the edges in apposition

by continuous hyperextension. When a few sutures of fine catgut or waxed silk have been inserted into the skin edges, the wound is sealed with a single layer of gauze soaked in collodion.

A sterilized plaster tube is now slipped over the finger with care to maintain the hyperextension, and the hand is dipped in sterile water. When the excess water has been squeezed from the plaster in the surgeon's hand, the patient is given control



FIG 323.—The incision.

and holds the finger in the desired position till the plaster is set. The sutures need not be removed until the plaster case is discarded at the end of five weeks.

I am deeply indebted to Mr. T. McW. Millar and Mr. D. Stewart Middleton, of the Surgical Out-patient Department of the Royal Infirmary, Edinburgh, both for the kindness they have shown in permitting me to treat their cases and for their sustained interest and advice.

BIBLIOGRAPHY

- MASON, MICHAEL L., *Surg. Gynecol. and Obst.*, 1930, 1, 611.
PLATT, HARRY, *Brit. Med. Jour.*, 1931, i, 611.
WILLIAMS, GWYNNE, *Minor Surgery and Bandaging*, 20th ed., 89.

BLOOD-PRESSURE DURING SPINAL ANÆSTHESIA

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THE article by Miss Joan Walker in the July issue of the BRITISH JOURNAL OF SURGERY on the use of hypertonic saline for the purpose of raising the blood-pressure, tempts me to record many facts and experiences similar to those she has noted, but to offer a different interpretation of those facts, and a different treatment for the conditions met with—a treatment, moreover, which has had a considerable measure of success. This is not in any way to decry the treatment of low blood-pressure by hypertonic saline, of which I have had no experience, but merely to show that many cases of low blood-pressure need no special treatment, that low blood-pressure and 'shock' are by no means synonymous terms, and that low blood-pressure when it requires treatment can frequently be remedied by other and somewhat less heroic measures.

Confining our attention for the moment to abdominal sections performed under spinal anæsthesia, one is struck by the fact that they divide themselves into two very definite and distinct groups: (1) Cases in which the blood-pressure falls at once and steeply before the peritoneum is opened; and (2) Cases in which the blood-pressure either rises or remains steady after the preliminary ephedrine and spinal anæsthetic have been given, and usually but not always falls when intra-abdominal manipulation such as exploration or the packing of gauze is undertaken.

The next point to notice is that the cases that show a steep and immediate fall of blood-pressure when the spinal anæsthetic is given are the acute abdominal conditions with great pain. So typical is this movement of the blood-pressure in these acute cases that if it does not occur it is safe to say that the diagnosis is mistaken and that the abdominal condition is not very acute. A few days ago I was asked to give a spinal anæsthetic to a man of 52 diagnosed as a perforated duodenum. On giving the spinal anæsthetic (anæsthesia to the nipple line) the blood-pressure fell, but only 10 mm. Hg—from 120 to 110. Very thorough and persevering efforts were made to find the perforation, during which the blood-pressure fell to 75 mm. Hg, rising to 100 within five minutes of their cessation. No perforation was found, but a partial obstruction by a band of the terminal ileum. During these proceedings I ventured to suggest to the surgeon that no perforation would be found, and, further, that the abdominal condition, whatever it was, was not very acute. The actual findings were entirely in accordance with the movements of the blood-pressure. *Table I* gives the records of 20 acute abdomens opened under spinal anæsthesia.

The 20 cases have been selected from my records; they are by no means consecutive. It is pertinent to ask why select the cases if the fall of blood-pressure is so characteristic of acute abdominal conditions. The reasons are two in number. In the first place I have tried to give as great a variety of acute cases as possible, and in the second place I have tried to choose cases uncomplicated as far as possible by other conditions likely to cause a sharp fall of blood-pressure.

Table 1.—BLOOD-PRESSURE RECORDS IN 20 OPERATIONS PERFORMED UNDER SPINAL ANÆSTHESIA FOR ACUTE ABDOMINAL CONDITIONS

No.	INITIALS	SEX	AGE	DISEASE	B.P. 1	B.P. 2	B.P. 3	CONDITION FOUND AND REMARKS	RESULT
1	A. R.	F.	50	I.O.	110	70	45-50	Two feet gangrenous gut removed	Recovery
2	W.	F.	40	T.O.	100	?	90-100		Recovery
3	W.	M.	54	I.O.	160	85	85-140-115	Prolapse of gut (3 ft.), foramen of Winslow	Recovery
4	D.	M.	70	I.O.	140	60	60-90	Mesenteric thrombosis (2½ ft. gut)	Died (10 days)
5	E. H.	F.	—	I.O.	170	100	90-140		Died (3 weeks)
6	H. D.	M.	—	P.D.	120	63	?	Perforation 12 hours old	Recovery
7	A. B.	M.	53	P.D.	140	75	80-65		Recovery
8	N.	F.	71	I.O.	145	90	90-112-100	71 years old. Very fat	Died (17 days)
9	A.	M.	—	A.A.	120	70	64-90	Very extensive peritonitis	Recovery
10	A. L.	M.	56	A.A.	120	?	60	Very extensive peritonitis	Recovery
11	E.	F.	44	I.O.	110	60	60-80		Died (16 days)
12	A. M.	M.	63	S.I.H.	150	80	70-92-85	Very ill. Bowel of doubtful vitality. Chronic bronchitis and emphysema	Died (4 days)
13	K. O.	F.	—	P.G.	120	?	?	2½ pints saline intravenously	Died next day
14	W.	F.	46	P.D.	120	60	60-60		Recovery
15	B.	F.	46	I.O.	110	?	?	Richter's hernia	Recovery
16	C.	F.	66	I.O.	140	?	?	66 years old	Recovery
17	B.	—	62	S.U.H.	180	?	?	Very great pain	Recovery
18	T. H.	M.	40	P.D.	130	75	60-120		Recovery
19	H.	F.	66	S.F.H.	120	75	64-78		Recovery
20	H. K.	F.	50	A.P.	180	70	70-60	Very ill	Died (4 days)

I.O. ... Intestinal obstruction
 P.D. ... Perforated duodenum
 T.O. ... Twisted ovarian pedicle
 A.A. ... Acute appendicitis
 S.I.H. ... Strangulated inguinal hernia
 P.G. ... Perforated gastric ulcer
 S.U.H. ... Strangulated umbilical hernia
 S.F.H. ... Strangulated femoral hernia
 A.P. ... Acute pancreatitis
 B.P. 1 ... Blood-pressure before spinal anæsthetic
 B.P. 2 ... Blood-pressure after spinal anæsthetic
 B.P. 3 ... Blood-pressure during operation

Amongst these other conditions that are frequently met with in the course of one's usual routine are the following: (1) Old age; (2) Anxiety or extreme nervousness; (3) Want of tone, as in the fat and flabby; (4) Overdosage with sedative drugs—paraldehyde, omnopon, and scopolamine; (5) A high blood-urea indicative of a toxic state; (6) The severely dehydrated. Any of these conditions, singly or in combination with each other or with an acute abdominal lesion, will cause a steep fall of blood-pressure within a few minutes of the exhibition of a spinal anæsthetic before the peritoneal cavity is opened. With the exception of (5) they are all fairly obvious and should not cause confusion. Still there are many patients who come with acute abdominal lesions who are neither fat nor flabby, nor severely dehydrated, nor overdosed with sedatives, who are still young and otherwise healthy, and not extremely nervous. It is in such cases that the steep fall of blood-pressure as the immediate result of a spinal anæsthetic is characteristic of an acute abdominal lesion.

The essential feature about all these cases is pain. Some were suffering from shock. Perhaps one ought to say that all were suffering from shock, but in such varying degrees that clinically there was the most marked difference between *Case 4*, an old man with mesenteric thrombosis, and *Case 9*, a man of 45 in comparatively good condition.

The next point to note is that the relief of pain and the fall of blood-pressure occurred simultaneously, both dramatic events. The conclusion is almost irresistible that the spinal anæsthetic caused both, and by the same action. There is no doubt that the pain is relieved by the action of the spinal anæsthetic on the posterior roots, and it seems almost certain that whether pain and pressor impulses are the same and carried by the same fibres or not, both are carried in the posterior roots and are paralysed by the spinal anæsthetic. The vasomotor centre which has been bombarded by these pressor impulses suddenly ceases to receive them and so ceases to send out vasoconstrictor impulses, hence the fall of blood-pressure. Parsons and Gray, in 1912, pointed out that pressor impulses leading to a rise of blood-pressure were productive of the first stage of shock, and it is now well known that the viscera of animals dying from shock are pale. The intense vasoconstriction is obviously a protective mechanism for the supply of vital organs with whatever blood the organism still possesses. It is important when the spinal anæsthetic has abolished this protective mechanism that the patient should be put in a slight Trendelenburg position so that the heart shall receive an adequate supply of blood. The action of the spinal anæsthetic in causing a fall of blood-pressure is too quick to be due to paralysis of the anterior roots (vasoconstrictors) and the exudation of fluid from the vessels into the tissue spaces. One must suppose that this latter process takes some time. Holt and Macdonald found that in order to reduce their animals to a state of shock they had to lose on an average no less than 48 per cent of their calculated circulating blood-volume. It is impossible to suppose that a spinal anæsthetic can in five or ten minutes cause any comparable loss; yet if spinal anaesthesia be the cause of shock, since shock is proportional to fluid loss, this is what one must suppose.

Not only does the rapidity with which the blood-pressure falls and its coincidence with the disappearance of the pain point to the action of the spinal anaesthesia on the posterior roots as being the cause of these phenomena, but experimental evidence supports this view. If a cat be given a spinal anaesthesia

nearly but not quite up to the origin of the phrenic nerves, the blood-pressure will fall to about 60 mm. Hg. If now the abdomen be opened and traction made on the stomach, the blood-pressure will fall still further, but resumes its former level on releasing the stomach. If this traction be repeated at intervals of a few minutes it will be found that each succeeding fall is followed by a rise of blood-pressure, and that this rise becomes more marked on each occasion. It is obvious that pressor impulses which had been abolished by the spinal anæsthesia are gradually coming into play again as the anæsthesia disappears. That the anæsthesia is disappearing is proved by the fact that the base line of 60 mm. Hg to which the spinal anæsthesia had originally reduced the blood-pressure, has been gradually rising throughout the experiment.

If then we take this view of the fall of blood-pressure—namely, that it is due to the abolition of pressor impulses by the spinal anæsthesia—we shall not call it 'shock', we shall not suppose it to be due to the leakage of plasma from the circulation, and there will be no indication that this fall of blood-pressure needs any treatment at all. Whether it be desirable to institute any special treatment at the moment or not will depend entirely on the condition of the patient before the spinal anæsthesia was given. If, for example, the patient has a recent (2-3 hours) duodenal perforation, when he comes on to the operating table he will be suffering intense pain in spite of the morphia which he has in all probability received. In such a case the spinal anæsthesia will give him such tremendous and almost instantaneous relief that his condition is much improved in spite of having lost half his blood-pressure. The patient at least thinks so. No treatment is necessary.

If on the contrary the patient be severely dehydrated from any cause, then either prior to or immediately after the spinal anæsthesia he will require treatment to restore his diminished blood-volume. One such case came on to the table with a blood-pressure of only 70 mm. Hg. The spinal anæsthesia caused the radial pulse to disappear. Intravenous saline (3 pints) given slowly throughout the operation, together with the intramuscular injection of pituitrin, raised the blood-pressure to 130 mm. Hg by the time the operation was finished, and the patient made an excellent recovery. The essential state to treat here was *lack of blood*, not lack of blood-pressure, which is primarily significant as an indication of the lack of circulating fluid. It is difficult to suppose that hypertonic saline would have been a *more efficient remedy in this case*, and yet it is just the kind of case in which one would imagine hypertonic saline would show to great advantage.

A third point to note is that in none of these cases is the blood-pressure low before the spinal anæsthetic is given. We so often hear that the blood-pressure is low in shock that we overlook the fact that in the early stages of shock the blood-pressure is high, and that it fails in spite of a high degree of vasoconstriction only when the patient or animal has lost between 40 and 50 per cent of his blood-volume. It is obvious that in the above list no such loss had taken place. The case just referred to might possibly have done so, as his blood-pressure was only 70 mm. Hg.

For the sake of comparison, *Table II* shows a series of 20 cases of non-acute abdominal sections done under spinal anæsthesia. All these cases, like the preceding group, had 100 mg. ephedrine injected with the local anæsthetic before the spinal anæsthetic. The first ten were all done by the same surgeon, and the second ten by ten different surgeons. It will be noticed that the blood-pressure rose in 18 out of the 20, and in the other 2 remained the same, *after the injection of the spinal*

anæsthetic, before the peritoneum was opened. Subsequently, during the operation it fell in every case. Here then is a totally different picture of the course of the blood-pressure which calls for a different explanation of the fall. If spinal anæsthesia causes first a stagnation of blood so complete as to set aside a considerable fraction from the circulation, and secondly an exudation of plasma from the capillaries comparable to that seen in traumatic shock, why does it not occur in the chronic case as in the acute? I can see no answer to that question. It is certainly not due to the condition of the acute case being more serious; it may or may not be. No one would suggest that a perforated duodenum in an otherwise healthy man, for instance, was as serious as a malignant growth of the colon or the head of the pancreas, or even a condition requiring gastrectomy. The reason why we get a fall in the acute case we have already shown to be the obliteration of pressor impulses by the spinal anæsthetic. In the chronic case, on the other hand, we do not get a fall of blood-pressure because the vasoconstrictors of the upper dorsal anterior roots are not touched by the anæsthetic, but are, on the contrary, powerfully stimulated both by the ephedrine and the condition of the patient. This overaction of the upper dorsal vasoconstrictors is simply part of the means that every organism uses to adapt itself to new conditions. It does not occur in the very old, the very feeble, or the very ill, and in these cases the blood-pressure falls.

Table II.—BLOOD-PRESSURE RECORDS IN 20 OPERATIONS PERFORMED UNDER SPINAL ANÆSTHESIA FOR NON-ACUTE ABDOMINAL CONDITIONS

NO.	INITIALS	SEX	AGE	OPERATION	B.P. 1	B.P. 2	B.P. 3
1	A. F.	M.	—	Colostomy and excision ..	130	150	100
2	A. K.	M.	56	Gastro-cholecystostomy ..	134	138	60
3	D. D.	M.	65	Resection of growth of colon ..	150	150	65
4	B. O.	F.	45	Double lateral anastomosis ..	100	140	90
5	C. G.	M.	60	Gastro-enterostomy ..	130	130	60
6	C. B.	F.	—	Resection of growth ..	110	150	80
7	D. B.	F.	—	Appendix (interval) ..	118	123	85
8	E. R.	F.	68	Resection of growth ..	145	160	80
9	G. S.	M.	49	Gall-bladder.. ..	120	160	125
10	H. M.	F.	—	Gall-bladder.. ..	130	135	60
11	J. H.	M.	37	Gastric ligation ..	120	160	120
12	K. W.	M.	48	Resection of growth ..	120	170	80
13	M. L.	M.	35	Gastrectomy.. ..	110	150	90
14	M. R.	M.	37	Nephrectomy ..	145	190	102
15	N. S.	M.	56	Gastro-enterostomy ..	115	130	75
16	O. A.	F.	43	Double pyosalpinx ..	120	140	110
17	P. L.	F.	72	Exploration for malignancy ..	140	150	60
18	R. N.	F.	40	Transverse colostomy ..	130	140	110
19	J. A.	M.	48	Appendix (interval) ..	140	170	125
20	— S.	F.	48	Cholecystectomy ..	125	145	80

B.P. 1 = Blood-pressure before spinal anæsthetic

B.P. 2 = Blood-pressure after spinal anæsthetic

B.P. 3 = Blood-pressure after opening of peritoneal cavity

Why does the fall occur later on? The time at which it occurs—namely, when the exploration is made, or severe traction is made, or large swabs are being packed into the abdominal cavity—suggests very strongly that it is due to stimulation of the vagus, a widely distributed nerve untouched by the anæsthetic. The fact

that a fall of blood-pressure is more likely if the operation be on the upper abdomen than on the lower points in the same direction, as does also the animal experiment already referred to in this paper.

Miss Walker refers to the idea that the fall of blood-pressure is due to absorption of the anæsthetic into the blood-stream, but rejects it as unlikely. I agree with her, but should put it more strongly. Ten milligrams of novocain injected into the cisterna magna of a cat will kill it in one and a half minutes by paralysis of the respiratory centre. If artificial respiration be applied in time the cat will be rescued, and in a few minutes will be able to breathe by itself. If now we allow it an hour in which to destroy any novocain left in its system and then inject 20 mg. of novocain into the jugular vein, it affects neither the blood-pressure nor the respiration. Moreover in one experiment which I did with Macdonald we injected $7\frac{1}{2}$ mg. of novocain every minute for twenty-four minutes before the respiration was affected—that is, eighteen times the dose that was lethal if put into the cisterna magna. Again, I injected 300 mg. into the median cephalic vein of a patient in the course of ten minutes; the blood-pressure remained steady between 140 and 160 mm. Hg, and the respiration was unaffected. These facts have been published before; but one constantly sees this bog of blood absorption of a spinal anæsthetic treated as a serious danger.

One other point calls for notice, this time a difference in facts observed. Miss Walker records a fall of blood-pressure in 157 cases out of 300 (52·3 per cent), and 42 failures to obtain anæsthesia (14 per cent). Excluding acute cases, and patients in whom one would be unlikely to find vigorous adaptation to new conditions—that is, the fat and flabby, the very old, or very ill—I find that in the last 100 cases of which I have records, in only 5 (5 per cent) has there been a fall of blood-pressure before the operation began, and there have been no failures to obtain the required anæsthesia and muscular relaxation. It is perhaps impossible to explain such different results, but one factor may account for part of the discrepancy. I have used the following solutions—heavy duracain (a 10 per cent novocain solution), heavy percain (a 0·5 per cent solution), and heavy decicain (a 1 per cent solution), each with a specific gravity of 1·025. These solutions are injected with the patient lying on his side, and he is turned on to his back immediately after the injection is finished. In this way more posterior roots are affected than anterior, and as it is the paralysis of the anterior roots that causes a fall of blood-pressure, I regard this technique as both more reliable and safer than the use of light solutions.

A fall of blood-pressure during spinal anæsthesia may be due to one or more of many causes. Treatment, if any, will depend on the diagnosis of the cause. Whether any of these causes is more efficiently remedied by the use of hypertonic saline, which has certain admitted disadvantages, than by more ordinary methods, seems doubtful.

PATHOLOGICAL FRACTURE OF THE HUMERUS COMPLICATING LATE SECONDARY SYPHILIS

BY H. JACKSON BURROWS, LONDON

CASE REPORT

THE events in the following case are described in the order in which they presented themselves to the surgeon, as they seem more instructive in this sequence than in strictly chronological order.

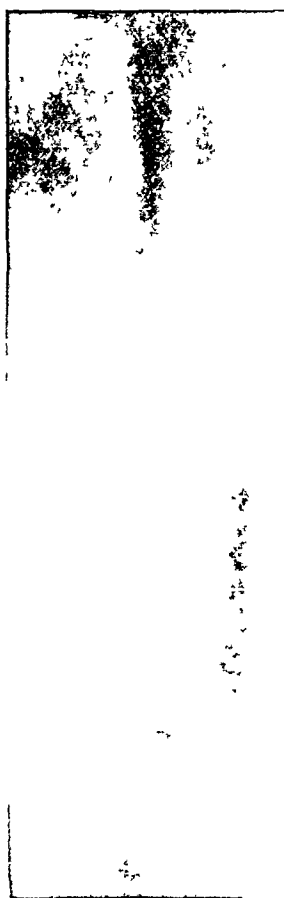


FIG 324 — The right humerus (posterior oblique projection), showing the pathological fracture.

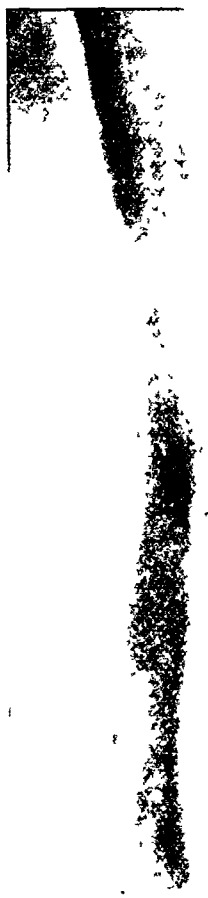


FIG 325 — Posterior projection of the fractured right humerus.



FIG. 326.—The left radius and ulna.

HISTORY GIVEN BY THE PATIENT AND CLINICAL EXAMINATION.—The patient was a maintenance mechanic, aged 28 years. Ten weeks previously, when throwing a skittle ball with

his right arm, he heard something snap, and the arm dropped and became useless. He was treated in a hospital with a Thomas's arm bed-splint. After seven weeks he returned to work.

The day before he came under observation, he was boarding a tramcar, which started suddenly as he was hanging on with his right hand, when he experienced sudden pain in the right arm and loss of use of the limb.

On examination, any movement of the right arm resulted in pain.

X-RAY EXAMINATION.—The humerus of each side was X-rayed. The right showed an oblique fracture just below the middle (*Figs. 324, 325*). Both showed subperiosteal new bone

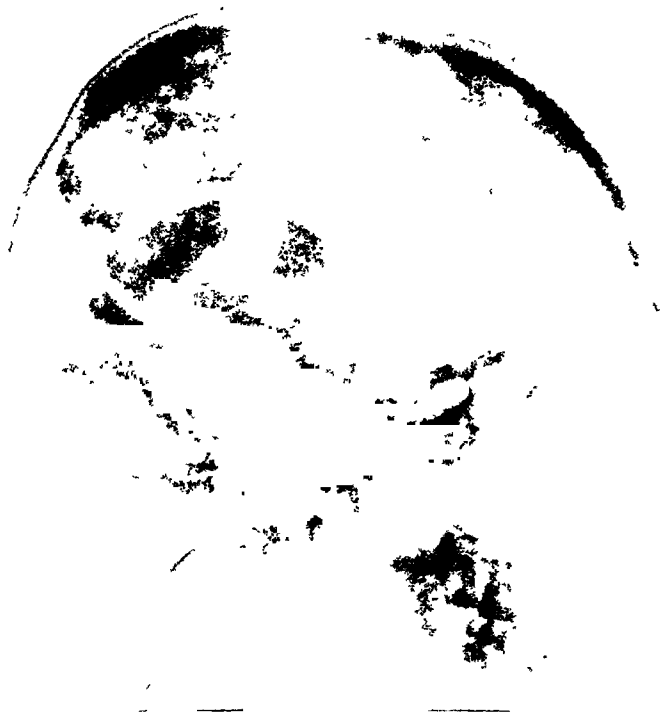


FIG. 327.—The skull.

formation which blended with the cortex; this was irregularly rarefied and showed a trabecular structure very clearly. The eccentric thickening of the cortex and the clear delineation of its texture were somewhat reminiscent of the appearances in *Paget's disease*.

A screen examination of the whole patient was carried out, and radiographs were made from those bones which appeared affected when so viewed. These comprised all the bones of the body except the pelvis, skull, hands, and feet. The changes were similar to those described above, except that localized patches of decalcification were more pronounced in some situations—left radius and ulna (*Fig. 326*), both fibulæ.

PROVISIONAL DIAGNOSIS.—Syphilis was considered the most likely cause of the generalized bone condition associated with the pathological fracture, although the patient was old to be giving the first indications of congenital disease, of which stigmata were absent, and seemed young for the bony manifestations of acquired disease. The most probable differential

diagnosis was considered to be generalized fibrosis of bone ('osteitis fibrosa' without hyperparathyroidism), or possibly hyperparathyroidism.

FURTHER INVESTIGATIONS.—

Urine.—Bence-Jones albumose was absent.

Blood Wassermann Reaction.—This was negative on three occasions, the last after a provocative dose of N.A.B. (0.45 g.).

Further X-ray Examination.—Radiographs were made from those parts of the skeleton which had been examined previously on the screen only (e.g., Fig. 327). All showed the changes described, and they were particularly beautifully seen in the hands and feet.

Blood Chemistry (Dr. H. E. Archer, Dr. G. A. Harrison, and Dr. William Smith).—

Serum calcium—

13.0 mg. per 100 c.c. serum (1st exam.).

11.3 mg. per 100 c.c. serum (2nd exam.).

Inorganic phosphorus—

3.7 mg. per 100 c.c. plasma (1st exam.).

3.4 mg. per 100 c.c. plasma (2nd exam.).

Plasma phosphatase 0.788 units.

FINAL DIAGNOSIS.—Although nothing abnormal had been reported in the screen examination of the pelvis, the radiograph clearly showed the tracks of intramuscular injections. When confronted with these the patient admitted to having received treatment for acquired syphilis at another hospital.* He had not previously admitted to any illnesses, but had not been very closely questioned. In this connection it is worth recording that he was prosecuting a claim for compensation for his accident against the tramway undertaking.

PROGRESS OF FRACTURE.—A Robert Jones humerus extension splint was applied. At the end of four weeks clinical examination revealed union, and active movements of shoulder and elbow were started. Full function was regained.

PROGRESS OF GENERAL OSSEOUS CONDITION.—Seventeen months after the first examination fresh skiagrams were taken of the bones of each arm (Fig. 328), forearm, hand, thigh, and leg. They had returned to an almost normal condition.

The patient stated that he had not received any further treatment.

COMMENTS

The features of particular interest in this case are the following:—

1. The onset of generalized osseous changes in spite of intensive treatment, which had started in the primary stage and was still being carried out when the pathological fracture occurred, and which had resulted in a negative Wassermann reaction.

2. The secondary character of the condition, as shown by its early onset, by its generalized distribution throughout the skeleton, and, if the patient's statement is accepted, by its spontaneous resolution.



FIG. 328.—The right humerus after an interval of seventeen months from the date of Fig. 325.

* I am much indebted to Dr. V. E. Lloyd for the following information:—

Fifteen months previously, first attendance with primary penile lesion (*Sp. pallida* present) and inguinal adenitis. Wassermann ++. No secondary signs. Treatment with arsenobenzole and bismuth injections. Wassermann negative prior to second course of treatment, starting six months after the first. Wassermann negative also at end of third course of treatment fourteen months after the onset. Total medication in fourteen months: N.A.B. 12.85 g.; bismuth oxychloride, twenty-seven injections of 2 c.c.

3. The complication of a pathological fracture, and the readiness with which this united.
4. The circuitous method of arriving at a diagnosis.
5. The medico-legal implications.
6. The possibility that the generalized bone changes were the result of therapy and not the result of the disease.

In addition to the acknowledgements made in the text I have to thank Mr. R. C. Elmslie for taking the patient under his care for certain of the investigations ; and Dr. J. Wylie, Dr. D. M. Stone, and Dr. G. T. Loughborough for permission to reproduce the skiagrams.

A COMPARISON OF THE RESULTS OF SPINAL FIXATION OPERATIONS AND NON-OPERATIVE TREATMENT IN POTT'S DISEASE IN ADULTS

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EVIDENCE, both clinical and pathological, is in favour of the view that the tubercle bacillus in a 'cured' lesion is not destroyed by the body; it is only encapsulated by the reactive changes of the host and is thereby rendered more or less innocuous. This conception of the disease is probably not invariably true, but for practical purposes it must be held that a tuberculous focus always carries the danger of containing live tubercle bacilli.

In order, therefore, to obtain a permanent cure of the disease, excision should be very satisfactory; but actually its application is limited because, firstly, general treatment is always required as well as treatment for the local lesion; secondly, excision is frequently not complete; and thirdly, some lesions are very difficult of access. In early tuberculous lesions of bone before joint involvement has occurred, excision of the focus is often advocated, and is probably a sound line of treatment; but in the case of the spine, with a few heroic exceptions, it is never attempted. Consequently, until some specific remedy is introduced, we are left with 'cure' by conservative treatment, which means encapsulation of the focus by fibrous tissue. This is a fairly satisfactory result except under two conditions, one where a communication with a blood-vessel persists and metastatic lesions occur repeatedly, and the other where movements take place (i.e., in joints) which are liable to cause liberation of the bacilli and reactivity of the disease. The former accident is as a rule beyond our control, but the latter can be obviated to some extent by treatment. It is this decrease in the danger of reactivity that has, generally speaking, caused the absolutely fixed joint to become recognized as the best end-result of tuberculous disease.

As the absolutely fixed joint (true bony ankylosis) is very seldom achieved in cases of pure tuberculous infection, artificial fixation of the joint was suggested, and at present plays a big part in the treatment of the condition. The problem of the stage at which the operation should be performed is one which requires very careful consideration, and the main object of this article is to help to establish the place of fixation operations in spinal disease in adults.

The idea of absolute immobilization afforded by operative aid, the short-cutting of the natural healing process, the essentially conservative nature of the operation, its relatively easy technique and low mortality-rate, all combined to render treatment by the creation of bony ankylosis very popular, especially in cases where the economic factor had to be considered. The lack of anterior consolidation induced by early fixation and the increased frequency of late abscess-formation were not appreciated until later.

The literature on this subject is (as is the way with all controversial subjects) relatively enormous, there being more than forty articles published during the last three years. A perusal of this and previous literature is useful in that it shows the cautious attitude adopted by the leading authorities on spinal tuberculosis in England, in contrast to the enthusiasm with which the operation was advocated in America.

Albee¹ first performed his operation in 1909 and Hibbs² in 1911. Later, in 1918, Hibbs reported a series of 210 cases without any primary mortality and with only 4 cases in which pseudo-arthritis formed subsequently. This was a very satisfactory report as far as immediate results were concerned, and in view of this as well as of the other appealing factors in favour of the operation, the American enthusiasm was sustained for many years, and even as late as 1935 the operation was advised on any patient at any age, provided the surgical risk was good. That there were limits to the indications for the operation, however, has been expressed on many occasions, and the general idea of these may be obtained from the following quotations from the literature.

In 1923 Girdlestone,³ concluding an analysis of 100 cases, stated: "In adults posterior spinal fixation is reliable and has great value as part of the conservative treatment of Pott's disease." Gauvain,⁴ in 1933, is of the opinion that "in adults their employment is more often justifiable, but I still hold the view that they are not indicated when the disease is active, and that, in other words, their value is rather in the nature of effective internal splintage to replace ineffective external splintage". King,⁵ in 1933, reporting on the results of 93 cases that had had spine fusion operations, makes these interesting observations: "The stability of the kyphos is mainly dependent upon adequate anterior vertebral-body contact. Correction obtained by abduction of its jaws causes instability by producing a dead space in the diseased area. Posterior spinal fusion does not restore sufficient stability to maintain correction obtained in this manner". Calvé,⁶ in 1935, advocated that it should only be performed when the disease is in the stage of an end-result, and this may be taken as a summary of the present-day opinion of 'when to operate'.

The series of cases analysed below supports this view in general, and not only confirms the fact that the stability of a kyphos is mainly dependent on adequate anterior vertebral-body contact, but also the results indicate that posterior fixation by operation in the early stage of the disease delays anterior fixation between the vertebral bodies (apparently because it prevents approximation in the later stages of the healing process), and, as a result of this lack of consolidation, abscess formation and reactivity are more common in the cases treated by operation.

CHOICE OF CASES FOR COMPARISON

A comparison of the results of 100 cases treated by non-operative means and 50 cases treated by fixation operations has been made, together with an attempt to obtain cases of the greatest comparative value. For this purpose those with the following factors in common have been chosen, but beyond this there has been no selection of cases.

Age.—All cases were of sixteen years of age or older at the commencement of the disease.

Site.—All the lesions were in the dorsal or lumbar regions.

Stage of Evolution of the Disease.—All the cases were in an early stage of the initial activity at the commencement of the treatment, so that cases with a hopeless prognosis on admission because of old-standing disease, incorrect previous treatment, or lack of treatment altogether are not included.

General Treatment.—All cases have had the same general treatment at the same sanatorium, but whereas in those treated by operation it has been for twelve months following the fixation, in those without operation it has been continued for a period of eighteen months on the average.

CLASSIFICATION OF CASES

The comparative value of the cases is still further increased by a radiological classification of the initial lesion, a radiological classification of the end-result, a clinical classification of the end-result, and an efficient follow-up scheme. The initial radiological classification shows the extent of the disease in the two groups of cases at the commencement of treatment, and indicates that on the whole a rather more extensive type of lesion has been dealt with by operation, but the differences are not sufficient to prevent a reasonable comparison, as given in *Table I*.

Table I.—EXTENT OF DISEASE IN OPERATION AND NON-OPERATION CASES AT COMMENCEMENT OF TREATMENT (INITIAL RADIOLOGICAL CLASSIFICATION)

CLASS OF LESION	OPERATION CASES	NON-OPERATION CASES
	per cent	per cent
1. Minimal lesion.. ..	8	17
2. Metaphysial and osteomyelitic of two vertebral bodies	66	73
3. Similar to (2) but involving more than two vertebral bodies ..	26	10

Classification A: The Radiological Appearance of the Initial Lesion.—It is convenient to consider this on a basis of the degree of bony destruction. Three main classes may thus be formed:—

Class 1.—A minimal lesion, usually metaphysial⁷ (often called tuberculous epiphysitis), sometimes periosteal, or even both metaphysial and periosteal (*Fig. 329*).

Class 2.—A metaphysial and osteomyelitic lesion involving not more than two adjacent vertebral bodies.

Class 3.—Similar to *Class 2* but involving more than two vertebral bodies.

Classification B: The Radiological Appearance of the End-result.—This classification is made on a basis of the degree of fusion anteriorly between the vertebral bodies as seen on the X-ray films. The lesions must also show good surrounding density with a clear outline, thus indicating quiescence.

Class 1.—Definite complete fusion of the diseased area. The majority of these will probably be bony ankylosis (*Fig. 330*).

Class 2.—A minimal degree of incomplete fusion as evidenced by a linear gap at the site of the lesion. This type will correspond to firm fibrous ankylosis pathologically.



FIG. 329.—A *Class 1* initial lesion—i.e., minimal degree of involvement.



FIG. 330.—A *Class 1* result, same case as Fig. 329.



FIG. 331.—A posterior dislocation. This type of end-result is only seen in cases which have an onset in childhood.

Class 3.—A very definite gap (a quarter to half an inch) between the bones affected at the site of the lesion. This will correspond to insecure fibrous union pathologically.

Class 4.—An extensive loss of substance at the site of the lesion, giving rise to either a gap greater than the depth of one whole vertebral body or to a pathological dislocation.

It must be realized that some cases which started as minimal lesions may remain so localized that complete destruction of the cartilage does not occur. In these cases a small gap will be seen in the radiograph; this will not represent an area of cavitation, however, but merely the remains of an intervertebral disc. When *Class 1* and *Class 2* radiological end-results are considered together, as GOOD, as in *Table II* and as used in the comparison of the cases, an error due to the misinterpretation of the radiographs is avoided.

Classification C: The Clinical Condition of the End-result.

Class A.—Up and about, fit for full day, no symptoms.

Class B.—Alive and well, but only up part time, minor symptoms.

Class C.—Reactivity of the spinal disease, or the occurrence of any complication such as abscess formation, sinus formation, and paraplegia.

Class D.—Died.

The degree of deformity (kyphos) has not been considered in classifying the end-results of these cases. This is due to the fact that, firstly, the cases with the soundest fusion often show well-marked deformity; secondly, the gross deformities are seldom seen in cases which have not had an onset in childhood (*Fig. 331*), and so the question of degree of deformity is of minor significance in the adult, even from a cosmetic point of view.

In *Table III* only *Class A* clinical results are considered GOOD.

The Follow-up Scheme.—Under the London County Council all cases of a tuberculous nature are followed up for a minimum period of five years after they have returned to home conditions and provided that they remain quiescent. They are also as a rule supervised by the same medical officer who has had charge of them during their stay in hospital.

The length of time in which the cases comprising the clinical groups *A* and *B* have been under observation as quiescent and under home conditions is an average of five years for the operation group and three years for the non-operation group.

COMPARISON OF END-RESULTS

(*Tables II, III, IV*)

The end-results, in order to simplify comparison, may be summarized as follows:—

Non-operative Treatment.—Gives:—

a. 62 per cent GOOD radiological results—i.e., cases showing bony fusion or fibrous fusion with a minimal gap.

b. 70 per cent GOOD clinical results—i.e., cases which are back under home conditions, are fit for a full day of work, and have no symptoms.

c. 4 per cent of cases showing late abscess formation, i.e., after they had finished treatment and had been discharged.

Operative Treatment.—Gives:—

a. 38 per cent GOOD radiological results.

b. 32 per cent GOOD clinical results.

c. 40 per cent of cases showing late abscess formation.

These main results of the analysis are so striking and decisive that, even allowing for the slightly more extensive type of lesion dealt with and the less general treatment received by the operative group, the following definite conclusions can be formed :—

1. Cases treated by non-operative means give better anterior consolidation.
2. Cases treated by non-operative means give better clinical results.
3. Cases treated by non-operative means are much less liable to develop late abscess formation.

Table II.—COMPARISON OF END-RESULTS RADIOLOGICALLY

CLASS OF END-RESULT		OPERATION CASES	NON-OPERATION CASES
1. Bony ankylosis	} GOOD	per cent 8	per cent 10
2. Firm fibrous ankylosis		30	52
3. Insecure fibrous ankylosis	..	46	36
4. Large gap or dislocation	..	4	—
5. Remained active until death	..	12	2

Table III.—COMPARISON OF END-RESULTS CLINICALLY

CLINICAL CLASS			OPERATION CASES	NON-OPERATION CASES
A. Normal life.	GOOD	..	per cent 32	per cent 70
B. Restricted life	14	6
C. Reactivity	32	14
D. Died	22	10

Table IV.—CHIEF COMPLICATIONS AND TIME OF THEIR OCCURRENCE IN HISTORY OF DISEASE

COMPLICATION	OPERATION CASES			NON-OPERATION CASES		
	Before Operation	During After-treatment	After Discharge	Before Treatment	During Treatment	After Discharge
Abscess ..	per cent 26	per cent 10	per cent 40	per cent 54	per cent 1	per cent 4
Sinus ..	4	—	2	8		
Paraplegia ..	6	—	2			

FURTHER RELATIONSHIPS BETWEEN DIFFERENT ASPECTS OF SPINAL DISEASE, OF VALUE IN ASSESSMENT OF TREATMENT

It seems almost axiomatic to say that the firmer the anterior fusion the better the clinical result.

Table V definitely confirms this view, but perhaps not to such an extent as would be expected; this may, however, be due to the fact that bony ankylosis seldom occurs without secondary infection, and secondary infection is very prejudicial to a good clinical result. It must be clearly understood that "firm ankylosis, preferably bony, is the thing aimed at", as was enunciated by Percival Pott nearly 200 years ago.

The initial X-ray lesion has a very definite bearing on the X-ray end-result; this is shown by *Table VI*, in which it will be seen that in the cases treated by non-operative measures:—

Minimal lesions (*Class 1*) give 16 out of 17 results (94 per cent) of bony ankylosis or of minimal-gap fibrous ankylosis—i.e., the class of result which is considered GOOD from the radiological point of view.

Table V.—RELATION OF DEGREE OF ANTERIOR FUSION TO CLINICAL
RESULT IN NON-OPERATIVE GROUP

DEGREE OF ANTERIOR FUSION (X-RAY RESULT)	CLINICAL RESULT			
	<i>Class A</i>	<i>Class B</i>	<i>Class C</i>	<i>Class D</i>
Bony fusion (<i>Class 1</i>) ..	per cent 80	per cent 10	per cent 10	per cent —
Fibrous union with minimal gap (<i>Class 2</i>)	71·2	3·8	15·4	9·6
Fibrous union with moderate gap (<i>Class 3</i>)	69·5	8·4	13·4	8·4

Table VI.—RELATION OF RADIOLOGICAL LESION AT COMMENCEMENT OF TREATMENT
TO RADIOLOGICAL END-RESULT IN NON-OPERATIVE GROUP

INITIAL LESION	END-RESULT				
	<i>Class 1</i>	<i>Class 2</i>	<i>Class 3</i>	<i>Class 4</i>	Remained Active
Minimal lesions (<i>Class 1</i>) ..	3 cases	13 cases	1 case	—	—
Involving two vertebral bodies (<i>Class 2</i>)	7 cases	36 cases	28 cases	—	2 cases
Involving more than two verte- bral bodies (<i>Class 3</i>) ..	—	3 cases	7 cases	—	—

Lesions involving the bodies of only two vertebræ (*Class 2*) give 43 out of 73 cases (58·9 per cent) of GOOD results radiologically.

Lesions involving more than two vertebral bodies (*Class 3*) give only 3 out of 7 cases (43 per cent) of GOOD results.

A further significant point is that there are no *Class 4* end-results in the non-operative group (100 cases); the tendency of the disease to remain localized if correct treatment is instituted is thus well shown. When the onset of the disease is in childhood, however, *Class 4* end-results are relatively common (cases with gross degrees of deformity), in marked contrast to the adult form.

CONSERVATIVE TREATMENT

The length of time required by conservative treatment in adults is actually remarkably constant, the average being eighteen months, again a marked contrast to the more prolonged and highly variable period of treatment required when the onset is in childhood.

The method of treatment used is based on the pathological evolution of the disease as demonstrated by serial radiography and occasional pathological specimens. It may be summarized as follows:—

1. *To Obtain Quiescence.*—For this purpose immobilization in recumbency is required until the activity as demonstrated clinically and radiologically has subsided. A spinal frame or plaster-of-Paris bed is the method employed, and about twelve months is the period of time required. A moderate degree of hyperextension is used at this stage, just sufficient to create a state of absence of compression in the diseased area, and so mechanical destruction as far as possible is prevented.

2. *To Obtain Consolidation of the Diseased Area, when Activity has Subsided.*—This is carried out by firstly allowing the patient to be free in bed for about two months, and then allowing him to get up in a spinal brace, taking about three months before the full normal activities are resumed.

The degree of kyphosis is so slight, as a rule, in adults, that the production of secondary curves by extension above and below the diseased area is not required.

ILLUSTRATIVE CASES

The following case reports are of interest as illustrations in themselves of the main contentions of this article.

Case 1.—W. A. D., male, aged 22 years.

This case showed two lesions. The initial one of the eleventh and twelfth dorsal vertebræ was fixed by a graft extending from the spinous process of the tenth dorsal vertebræ to that of the first lumbar, the operation having been performed five months after the onset of symptoms. Post-operative treatment consisted of two months in a plaster-of-Paris bed; the patient was then allowed up wearing a spinal brace, and one month later commenced work as a saw-mill hand. This work was continued for two years, when the pain in the back recurred and radiological examination showed a second lesion between the eighth and ninth dorsal vertebræ.

The full conservative treatment was carried out for this second lesion, and the final X-ray film is very interesting. It is reproduced diagrammatically in *Fig. 332*, and shows that there is a definite gap (*Class 3* result) at the site of the initial lesion, but that the second lesion has consolidated well, no cavity remaining (*Class 2* result). On further examination of this film a slight degree of erosion of the anterior surface

of the tenth dorsal vertebra can be made out, and a faint shadow anterior to the affected region of the spinal column, suggestive of an abscess between the anterior common ligament and the vertebral body (aneurysm phenomenon). This probably indicates that excessive movement and strain at the site of the eighth and ninth dorsal vertebrae, as a result of the fixation of the spine below, were the factors that determined the level of the second lesion.

Case 2.—M. M., aged 37 years, female.

This case was a metaphysial and osteomyelitic lesion of the tenth and eleventh dorsal vertebrae, i.e., *Class 2*. It was grafted, while still active, about six months after the onset of the disease, the graft extending from the spinous process of the

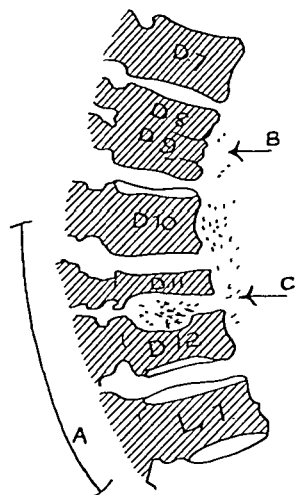


FIG. 332.—*Case 1.*—End-results of operative and non-operative treatment compared in the same case. Both show quiescence of the disease, but whereas there is good consolidation in the non-operative instance, a cavity is still present in the grafted lesion. A, Extent of graft—spine of D.10 to that of L.1; B, Second lesion, good density, clear outline and good consolidation—*Class 2* end-result; C, First lesion, good density, clear outline but cavity still present—*Class 3* end-result.

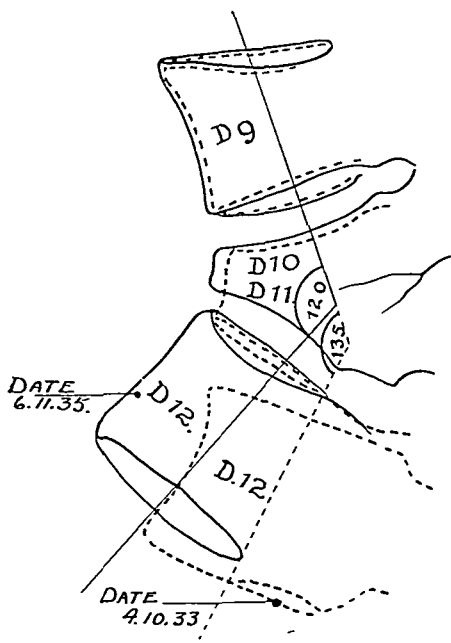


FIG. 333.—*Case 2.* Superimposed tracings of X-ray films showing 15° of collapse as a result of poor anterior consolidation, in spite of a graft.

eighth dorsal to that of the twelfth dorsal vertebra. After-treatment was prolonged, the total period of recumbency being fifteen months, because of abscess formation and paraplegia which arose six months after the operation. A comparison of the X-ray film immediately after grafting and the X-ray on discharge over two years later (*Fig. 333*) shows that further collapse of the diseased area has taken place.

This case illustrates the fact that an early graft will hold the diseased area sufficiently to prevent consolidation and yet will not be sufficiently strong as a sole means of support to prevent minor degrees of collapse from taking place.

Case 3.—G. A., aged 28 years, female.

This lesion of the twelfth dorsal and first lumbar vertebrae, of the metaphysial class, was grafted during the active stage of the disease and followed by full conservative treatment, giving a *Class 1* X-ray result (bony fusion). A few months after

discharge the patient felt something snap in her back, and a radiograph (*Fig. 334*) taken at this time revealed a fracture of the graft.

The following points are of special interest in this case :—

1. The graft was placed too high so that it did not span the diseased area (this occurred in one other case in the series of 50).
2. Full conservative treatment gave a *Class 1* result.
3. The strain on the portion of the spine unnecessarily fixed by the graft was sufficient to fracture the graft.

Case 4.—W. B., female, aged 36 years.

This case was grafted during the active stage of the disease, the graft extending from the spinous process of the eighth to that of the twelfth dorsal vertebra, the



FIG. 334—*Case 3.* A *Class 1* result. The graft spans the wrong spines, and also shows a fracture in its centre.



FIG. 335—*Case 4.* This shows extensive involvement of the lower dorsal region. The lowest lesion has a sequestrum in its centre, and is held open by a graft, while the upper lesion shows good consolidation.

lesion being a metaphysial and osteomyelitic one (*Class 2*) of the ninth and tenth dorsal vertebrae. After-treatment consisted of fourteen weeks' recumbency followed by a plaster-of-Paris jacket for four months, and then the patient was fitted with a spinal brace and recommenced her work as a machinist (sitting occupation).

After fifteen months under working conditions root-pains occurred, and the X rays showed a recurrence both above and below the original lesion. Further conservative treatment has now been employed for eighteen months, and the latest X-ray film (*Fig. 335*) shows that the extension of the disease was probably by way of the anterior abscess, the cupping of the anterior outline of the vertebral bodies being quite definite (aneurysm phenomenon), and the shadow representing the abscess anterior to the column fairly distinct. The lower lesion between the eleventh and twelfth dorsal vertebra is bridged by a graft and shows a wide cavity with sequestrum formation. The original lesion of the ninth and tenth dorsal

vertebræ shows fairly good consolidation. The upper lesion between the seventh and eighth dorsal vertebræ does not show the cavity formation like the lower one, because it has not been bridged and consequently held open by the graft.

PROGNOSIS

The prognosis in Pott's disease in adults is often quoted as being very grave, but this is not the case if correct conservative treatment is employed. *Table III* shows a mortality-rate of 10 per cent for 100 cases which had been under observation for an average period of six years since the onset of the disease. Actually the prognosis as regards life, length of treatment required, and end-result is much better in adults than in children.

A CONSIDERATION OF CERTAIN POINTS OFTEN PUT FORWARD IN FAVOUR OF OPERATION

The Economic Factor.—It is because of the economic factor in adults that operative fixation is so often advocated, but it is now agreed that general treatment for at least a year is required in all cases, and as conservative treatment only takes eighteen months the actual saving of time is only six months. But at what sacrifice is this six months saved? When it is realized that with operation there are only 32 per cent GOOD results, whereas with conservative treatment there are 70 per cent GOOD results, this economic factor as an indication for early operation cannot be taken into consideration.

The Immobilization Factor.—A spinal graft, after it has 'taken', together with recumbency, is the most effective means of obtaining immobilization; in fact it is the only way whereby movements during respiration can be completely prevented. This factor is the only unequivocal advantage of early grafting.

The Vascularizing Function of the Graft.—It has been suggested that the insertion of a tibial graft causes an increase of the vascularity of the diseased area of the spine (Albee) and so has a beneficial action on the course of the disease. The centre of a tuberculous lesion has an impoverished blood-supply, but the periphery has a zone of hyperæmia; it must be extremely difficult to decide that this zone of hyperæmia had been still further increased by a graft except possibly as a result of the minute study of a very large number of post-mortem examinations. However, allowing for such a possibility, the end-results of these cases suggest that there has either been no such beneficial influence, or, if there has, then it has been more than counteracted by the disadvantages of grafting during the early stage of the disease.

THE PLACE OF SPINAL FIXATION OPERATIONS IN THE TREATMENT OF POTT'S DISEASE

I. During the Active Stage.—As a result of this analysis it may be concluded that the operation of spinal fixation is contra-indicated in the active stage of Pott's disease in the adult, because :—

a. The clinical end-results are only half as good as those obtained in conservatively treated cases.

b. Anterior fusion between the vertebral bodies is delayed by posterior spinal fixation.

c. Posterior fixation is not sufficient to stabilize the spinal column, if there is poor anterior fusion.

d. The economic factor is put out of consideration because of the inferior end-results obtained by operation; further, if general treatment is used (and it should be used in all cases irrespective of local treatment) the saving of time is only six months.

In brief, the only object in performing the operation at all during the active stage is because of its immobilization effect, and this may be obtained sufficiently well by non-operative means.

2. In the Treatment of the End-result.—Spinal fusion used solely as a form of internal spinal support for the diseased area, when the optimum degree of anterior consolidation has been obtained, is much more efficient and much less cumbersome than any form of external appliance, and undoubtedly has a most useful place in establishing the permanent cure of the disease. A consideration of the radiological end-results obtained in this disease brings out the conclusions that:—

a. In the cases with bony ankylosis (*Class 1* type), where fusion is sound anteriorly, then probably nothing further is required; in fact, putting a graft into such a case is unnecessary (though it would be wiser to graft in a case where there was a doubt as to whether bony ankylosis had occurred or not).

b. Cases of fibrous union (*Classes 2, 3, 4*) are definite indications for grafting, but with the provision that in the case of a large loss of substance—i.e., *Class 4*—it must be a true end-result and must have been given many months, even years, to consolidate as much as possible anteriorly before the operation is carried out.

THE TYPE OF FIXATION OPERATION

There are several satisfactory methods of performing spinal fixation. An autogenous tibial graft combined with obliteration of the posterior articulations is probably the best, but what is most important is that it should immobilize the diseased area and the diseased area only. The reasons for advocating this short type of graft may be enumerated as follows:—

1. If the graft includes the spinous processes, posterior articulations, and laminae of the diseased vertebræ, then the actual area of disease will be immobilized efficiently—i.e., it is efficient as a form of spinal support for the weakened region.

2. When the disease has reached the stage of an end-result, there can be no object in throwing out of action normal intervertebral joints above and below the lesion. In fact, there is a positive objection to such a procedure, because the greater the length of spine immobilized the greater the stresses and strains on the rest of the column, if normal activities are undertaken, giving an increased liability to recurrence above and below the diseased area.

SUMMARY

The treatment of tuberculosis of the spine is discussed and the literature on fixation operations briefly reviewed.

A series of 100 cases treated by non-operative means and a series of 50 cases treated by operation are analysed and compared, chiefly in regard to their end-results.

The main conclusion arrived at is that spinal fixation operations performed during the active stage of Pott's disease prevent consolidation between the vertebral bodies at a later stage in the healing process ; and that consequently the place of operative aid is solely in the treatment of the end-result, in order to establish a permanent cure of the condition.

I am indebted to Dr. Colvin, of St. Luke's Hospital, Lowestoft, for suggesting the investigation.

REFERENCES

- ¹ ALBEE, F. H., " Spinal Tuberculosis ; Climatic and Operative Treatment ", *Amer. Jour. Surg.*, 1935, xxx. 60.
- ² HIBBS, R. A., " Treatment of Vertebral Tuberculosis by Fusion Operation ", *Jour. Amer. Med. Assoc.*, 1918, October.
- ³ GIRDLESTONE, G. R., " The Place of Operations for Spinal Fixation in the Treatment of Pott's Disease ", *Brit. Jour. Surg.*, 1923, x, 372.
- ⁴ GAUVAIN, Sir H., " Conservative Treatment of Surgical Tuberculosis ", *Recent Progress in Medicine and Surgery*, 1919-1933, 113.
- ⁵ KING, D., " The Results of Operative Fixation of Tuberculous Spines ", *Jour. Bone and Joint Surg.*, 1933, xv, 953.
- ⁶ CALVÉ, J., and GALLAND, M., " Osteosynthesis in Spinal Tuberculosis ", *Ibid.*, 1936, xviii, 46.
- ⁷ SEDDON, H. J., " The Morbid Anatomy of Caries of the Thoracic Spine in Relation to Treatment ", *Lancet*, 1935, ii, 385.

GASTROSCOPY

ITS HISTORY, TECHNIQUE, AND CLINICAL VALUE, WITH REPORT ON SIXTY CASES

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HISTORY

THE first attempt to view the interior of the stomach without having recourse to abdominal section was made by Kussmaul in 1868. The incandescent electric lamp had not at that time been invented, and Kussmaul's efforts were made by the rays of light from a naked flame reflected down a long straight wide tube. The clinical results were of no practical value at the time, but the attempt was an expression of the growing desire of clinicians to render visible the internal as well as the external surfaces of the body.

In 1871, Desormeaux laid down the general principles of endoscopy, and in 1876, just sixty years ago, the first indirect vision gastroscope was made by Nitze in Vienna. This instrument had an optical system similar to that of a modern cystoscope, with an electrically-heated incandescent platinum wire as the source of illumination. Difficulties were encountered with the very large diameter of the tube and the heat emitted by the platinum wire, and this instrument was only used experimentally on the cadaver. Mikulicz, however, interested himself in the problem, and in 1881 the first practical gastroscope was made under his direction. This instrument was narrower, and the glass enclosing the incandescent platinum wire was kept cool by a stream of water. In order to clear the projecting vertebral column the instrument was angulated through 30° near its distal end, with a prism at the angle to deflect the light rays to the eye-piece. Several patients were successfully examined with this instrument by Mikulicz with no fatalities, and in one case a carcinoma of the stomach was diagnosed by means of it. General anæsthesia was necessary for the instrumentation, which was described by Mikulicz as "an exquisite surgical manipulation which should only be undertaken by the most expert". Mikulicz, however, was disappointed with his experience. The view of the stomach was incomplete on account of the lack of a rotating nose-piece to the angulated instrument, so that a negative result from the examination was of no significance. In consideration of this and the necessity for general anæsthesia, Mikulicz held that exploratory laparotomy was to be preferred, and abandoned the method.

The effect of the practical failure of this very brilliant piece of work was to render clinicians sceptical of the possibilities of gastroscopy for the next thirty years. A number of unsuccessful attempts to solve the difficulties were made during this period. Rosenheim experimented with a straight rigid instrument, but found it too dangerous to use. Rewidzoff tried to make this safer by first introducing a flexible stomach tube and passing the rigid instrument through this protecting

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channel. It was found, however, that with a straight gastroscope the projecting lumbar vertebral column limited the intra-gastric field of vision considerably, and the results obtained were considered by the majority of practitioners insufficient to justify the discomfort entailed in the use of this rigid type of instrument.

In 1900 Kelling devised an instrument consisting of a number of hinge-jointed sections so that it could be introduced as a relatively flexible tube, but braced straight when in position by longitudinal tension wires. This instrument was angulated like that of Mikulicz to clear the vertebral column, but it also had the rotating nose-piece that Mikulicz had desired so much in order to see the whole area of the stomach. It was unfortunately too complicated and fragile, and was soon irreparably damaged before its use could be proved.

The solution of the problem was carried a stage further in this country by H. S. Souttar and Theodore Thompson in 1908. The experience of past workers together with a wide knowledge of physics and a remarkable degree of mechanical ingenuity enabled Souttar to construct an instrument which fulfilled all the physical requirements. It was angulated, carried a rotating nose-piece, and was constructed on the sectional principle of Kelling, so that it could be passed flexible and braced rigid for the examination. Souttar and Thompson successfully examined a number of cases at the London Hospital, which they reported in the *Quarterly Journal of Medicine*. The use of this instrument, however, was allowed to lapse for two reasons. In the first place, although a good view was obtained, general anæsthesia was necessary for the instrumentation, and so many cases subsequently had an operation that the anæsthetic for the gastroscopy was felt to be hardly justifiable. Also, radiological examination of the stomach by the barium meal, which was at that time coming into general use, seemed to satisfy all the requirements of gastric investigation. For the second time, therefore, gastroscopy was abandoned after it had been successfully practised.

As the limitations of radiology of the stomach became gradually recognized on the Continent, however, clinicians there turned again towards the ideal of gastroscopy and the problem of how to render it safe, reliable, and painless. The solution was found in a new optical system, in which a flexible telescope was made capable of transmitting an image notwithstanding a moderate degree of curvature of the tube. This was accomplished by the inclusion of a large number of lenses, each of which projected an image of small dimensions relative to the diameter of the lens. In 1932, under the direction of Schindler, a flexible gastroscope was made on this principle by Wolf, of Berlin, and this instrument has altered the whole aspect of the clinical value of endoscopic examination of the stomach. Schindler, and later Henning, of Leipzig, have shown that gastroscopy with the flexible instrument is absolutely safe, and that general anæsthesia is quite unnecessary for its satisfactory performance. With these two main difficulties removed, Henning has been able to show, as the result of an experience of over 2000 instrumentations, that gastroscopy is an examination of the greatest clinical value in the investigation of gastric cases.

THE INSTRUMENT

The instrument itself consists of a rigid upper part carrying the eye-piece, and a flexible lower part carrying the objective and the electric lamp. A sponge-rubber tip beyond the lamp facilitates the safe passage of the instrument and wipes the

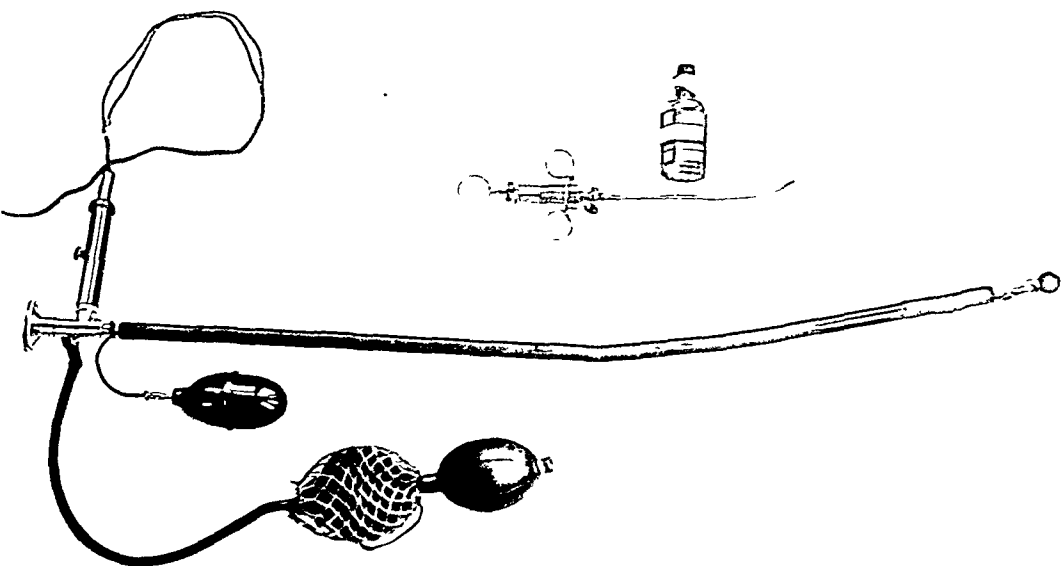


FIG. 336.—The Wolf-Schindler flexible gastroscope, with special sheath carrying the retracting balloon and inflating bulb. Above: The Brüning syringe for application of surface anæsthetic to the pharynx.

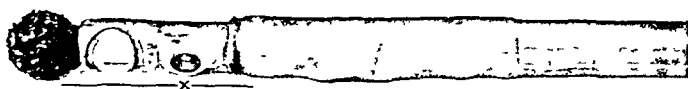


FIG. 337.—Distal end of instrument, showing sponge-rubber tip, lamp, window of objective, and balloon before inflation.

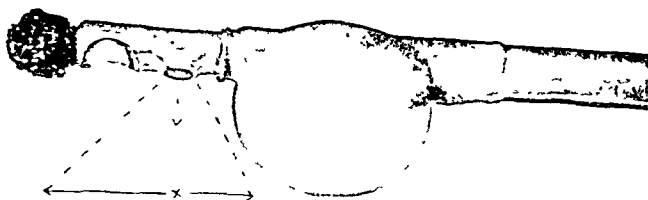


FIG. 338.—The same, showing retracting balloon inflated and lifting the objective away from the object X on the surface.

œsophagus in its descent, so that the optical window is not fouled by foreign matter. The upper end carries the electric connection and a rubber blower bulb whereby the stomach can be inflated with air during inspection. (*Figs. 336, 337, 338.*)

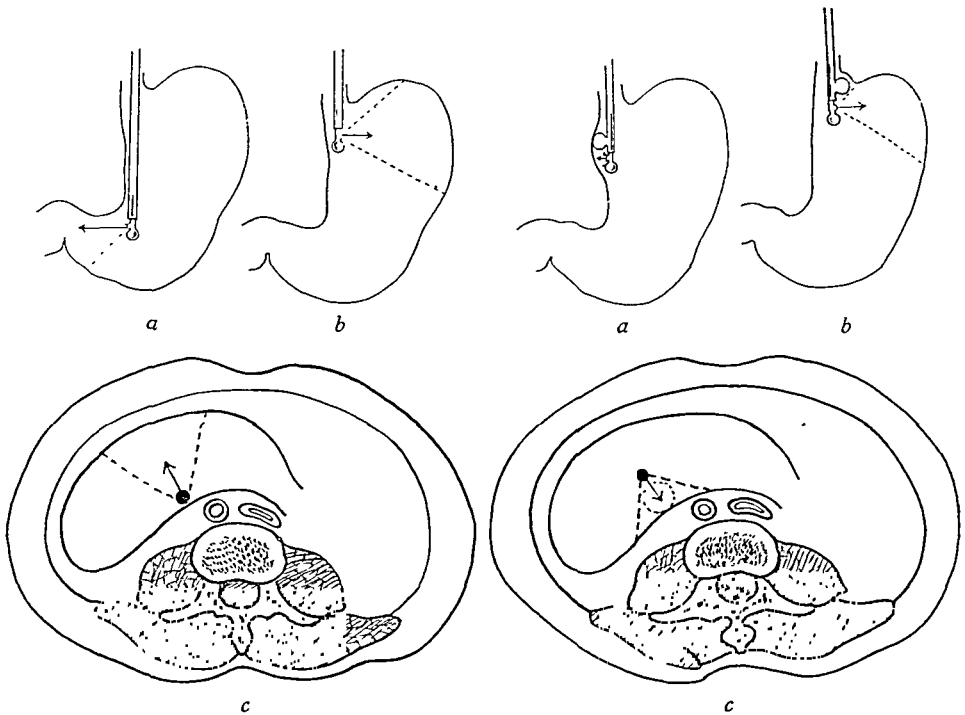


FIG. 339.—Diagrams showing gastroscopic views ordinarily obtained. *a*, Pylorus; *b*, Corpus; *c*, Anterior wall.

FIG. 340.—Diagrams showing views obtained with the aid of the balloon. *a*, Lesser curve; *b*, Fundus; *c*, Posterior wall.

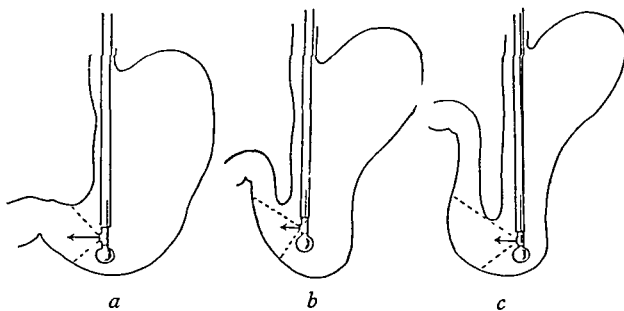


FIG. 341.—Variations in shape of stomach. *a*, Average; *b*, J-shaped; *c*, U-shaped.

The optical system of the Wolf-Schindler gastroscope imposes a limit of curvature of 34° deviation, beyond which it is not possible to see out of the objective of the instrument. Moreover, as this limit is approached, spherical and chromatic aberration render the view somewhat blurred. In practice, however, the gastroscope when in position is almost if not quite straight, and the view obtained is as sharply defined as that of the bladder on cystoscopy. Extreme degrees of flexion

of the instrument are required only for its safe introduction through the œsophagus. The 90° angle of view of the objective enables large areas of the interior of the stomach to be inspected in any one field, depending on the distance of the mucosa from the objective, and by rotating the instrument and withdrawing or advancing it in the stomach almost all of the gastric mucosa can be examined.

The position of the gastroscope in the body is as in the diagram (*Fig. 339*), rather to the left of the midline, with the objective in the corpus ventriculi. It will be seen that whereas the pyloric antrum and the greater curve of the body of the stomach lie well away from the objective and can be seen in true perspective, the upper part of the lesser curve and posterior wall of the stomach near the lesser curve are held close to the objective by the lumbar vertebral column and their proper examination presents difficulty on this account. This difficulty has been largely overcome by a modification devised by H. W. Rodgers and the author concurrently and made for us by the Genito-Urinary Manufacturing Co. This consists of a special rubber sheath designed to replace the standard sheath (*Figs. 337, 338*). It carries an inflatable rubber balloon, situated just proximal to the window of the objective and connected by a capillary rubber tube to a bulb at the upper end of the instrument. When the balloon is inflated by squeezing the bulb, it lifts the gastric mucosa out of contact of the objective and enables it to be satisfactorily inspected. *Fig. 340* show how this device allows a proper view to be obtained of the upper part of the lesser curve, of the posterior wall near the lesser curve—the site of the stoma of a posterior gastro-enterostomy—and of the lower end of the œsophagus, while by lifting up the fundal lip of the cardiac orifice as the instrument is partly withdrawn from the stomach, a better view of the fundus can be obtained.

In a visceroptic patient the sagging of the stomach may limit the gastroscopic field considerably. Henning has pointed out that although the L-shaped stomach drawn in *Figs. 339, 340* is that most frequently found, the viscus may be J-shaped or even U-shaped (*Fig. 341*). In the presence of a marked degree of ptosis, therefore, the dependent lesser curve may prevent a view of the pylorus from being obtained. This occurs in about 10 per cent of cases, and although it is undoubtedly a disadvantage when present, it need not necessarily prevent a satisfactory conclusion being drawn from the gastroscopy—i.e., when the suspected lesion is elsewhere than at the pylorus.

TECHNIQUE

The examination is preferably held in the morning, and except for a drink of water on waking, the patient is not allowed anything by mouth beforehand. In this way standard conditions of inspection of the gastric mucosa are obtained, when the stomach is empty and resting, and the epithelium is not stimulated to secrete by food or the thought of food.

The instrumentation can be carried out under local anæsthesia without any other pre-medication, so that it can be performed on an out-patient if necessary without undue discomfort. With a sensitive patient, however, and particularly for the first examination, it is the author's practice to give omnopon-scopolamine one and a half hours beforehand, and if necessary a second injection of morphia half an hour beforehand if the effect of the first is insufficient. It is important that the patient be not too drowsy, as his co-operation during the examination may be very helpful.

Surface anæsthesia of the mouth, pharynx, and œsophagus is obtained with 5 c.c. of 2 per cent decicaine or percaine solution to which five drops of 1-1000 adrenaline have been added. With the patient lying on his back, the mouth and pharynx are carefully painted all over with the solution—conveniently applied by a Brüning syringe (*see Fig. 336*), which keeps a wad of cotton-wool constantly wet with the liquid. It is necessary to take a full fifteen minutes over this stage, with three or four applications of anæsthetic, as complete desensitization of the pharyngeal mucosa is essential to a satisfactory examination. With proper anæsthesia, the stomach tube and gastroscope can be passed without the occurrence of any retching at all. First the lips, then the tongue, fauces, and oropharynx, are painted, and as the upper mucosa becomes insensitive, the laryngo-pharynx is anæsthetized, until on requesting the patient to swallow, the wool swab can be passed without spasm through the post-cricoid sphincter. It is important that the epiglottis and inner surface of the larynx should not be anæsthetized, as with an insensitive pharynx it may be difficult to prevent the stomach tube from entering the larynx, a mistake which cannot possibly occur if the larynx is still sensitive. It is not necessary to use a laryngeal mirror for the anæsthesia. The shape of the pharynx with the vertebræ lying posteriorly soon becomes familiar, and by keeping the swab always in contact with the posterior wall and lateral fossæ, no fluid can enter the larynx.

When fifteen minutes have elapsed since the commencement of the application of the local anæsthetic, a stomach tube is passed and the residual contents of the stomach are aspirated by a suction bottle. The character and quantity of the resting juice may here afford valuable evidence in regard to the case which may not have been present in the test-meal specimen. The patient is usually delighted to find that the stomach tube has been passed without any discomfort at all, and submits to the passage of the instrument with confidence.

Unlike cystoscopy, in which the cystoscope can be manipulated into any position inside the bladder, the gastroscope can only be rotated or moved in or out of the stomach; it cannot be moved sideways. Henning has therefore constructed a special table on which the patient can be moved, as it were, round the gastroscope, so that by tipping the patient upwards, downwards, or sideways, the stomach falls over in such a way that all parts of the interior can be brought into the field of the instrument. The author has adapted a table similar in principle to that of Henning, but he has found in practice that whereas the downward tilting of the table has often been of great use in examining the stomach, the sideways tipping of the table advocated by Henning has never enabled him to overcome a difficulty or to view an otherwise invisible area of mucosa. An ordinary operating table capable of being tilted downwards is therefore sufficient for the purpose, provided it has the necessary attachments. These consist of an adjustable head-rest, back-rest, foot-piece, and the knee-pillar invented by Henning. The author's table and the patient in position upon it are shown in *Figs. 342, 343*.

The patient lies on his left side with his back flat against the back-rest and his head moderately extended and slightly lower than the horizontal midline of the body. The upper thigh lies behind the knee-pillar and is held between it and the lower end of the back-rest, so that the patient cannot roll over. The lower knee is flexed in front of the knee-pillar and the foot is placed behind the foot-pillar, which is adjusted so that the limb is held firmly but comfortably. In this way the lower

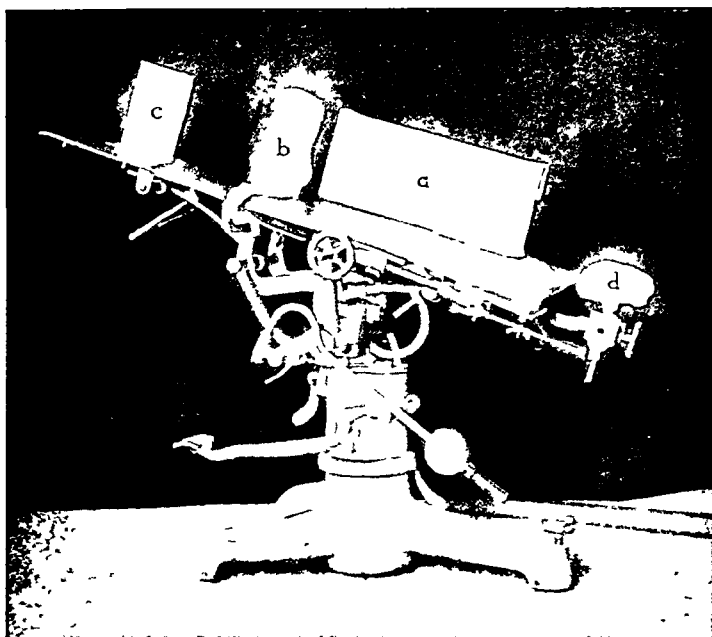


FIG. 342 —Operating table adapted for gastroscopy. Note (a) Back-rest, (b) Henning's knee-pillar (c) Foot-pillar, (d) Adjustable head-rest



FIG. 343 —Patient in position for gastroscopy. Note the position of the lower limbs. Note also the head between the hands of the assistant.

limbs prevent the patient from slipping down the table when it is tilted, and the patient's abdomen is dependent from the pelvis and free from pressure, so that the stomach within can move freely round the gastroscope under the influence of gravity. As a further support against slipping, the patient's left shoulder is in contact with the padded lower edge of the head-rest.

It is important to see that the patient does not arch his back, as this brings the lumbar vertebral column forward in the abdomen and adds greatly to the difficulty of the examination. A towel is now placed over the patient's face and the head is comfortably supported between the two hands of the assistant. With an assistant taking the weight of the eye-piece end of the gastroscope, two fingers of the



FIG 344—Illustrating the grip of the end of the instrument between fore and middle fingers of left hand. The end is flexed as the instrument is introduced into the pharynx.

surgeon's left hand are passed to the back of the patient's tongue and the end of the instrument is slipped in between them, gripped, and bent forward, so that the sponge-rubber tip passes down the pharynx without pressing unduly on its posterior wall (*Fig. 344*).

As the tip impinges on the post-cricoid sphincter, the patient is asked to swallow and the instrument passes easily to the cardia. A slight delay may be experienced as the end passes through the cardiac orifice, after which the instrument glides without resistance to the bottom of the stomach.

Apart from the avoidance of loose teeth, the passage of the gastroscope presents no special difficulties if the confidence of the patient is obtained, but there is one absolute rule that must never be broken. Under no circumstances nor at any stage of the instrumentation must any force be used. The end of the gastroscope is not pushed down, but rather swallowed naturally by the patient. Gastroscopy performed in this way is not only completely painless, but quite free from danger. If any

obstruction is encountered either above or within the stomach, then this is certainly due to an organic lesion, and no attempt should be made to push the instrument beyond it by force. Such obstructions might be pharyngeal or œsophageal pouches, an S-shaped lower end of œsophagus such as might occur with a symptomless cardio-spasm, or carcinoma of the upper part of the stomach, and under these circumstances (in practice extremely rare) the examination must be abandoned. With the gastroscope in position the patient should be able to lie quietly without pain or distress, beyond the discomfort caused by the displacement of the larynx to one side by the pressure of the rigid part of the instrument. He cannot of course articulate, but he can phonate, and he is able to indicate any untoward sensation to the operator.

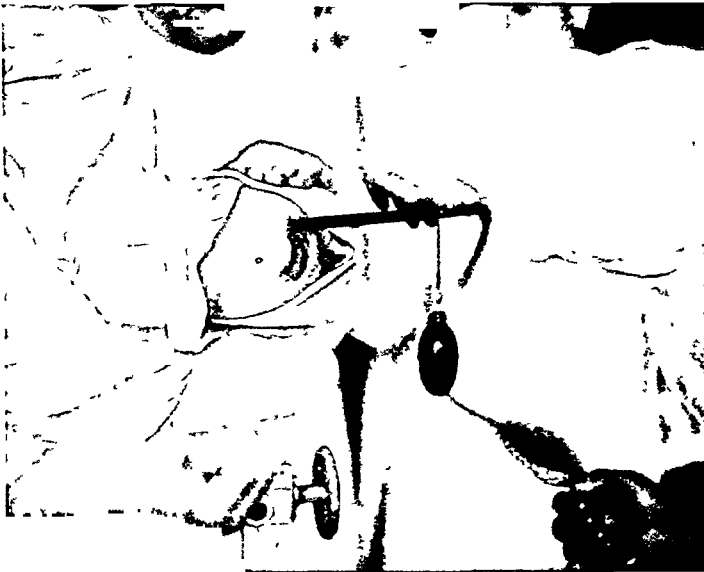


FIG. 345—Illustrating the position of patient, assistant, and operator during the examination. Note the instrument emerging from the right-hand side of the mouth.

Special care is taken to see that the patient's upper lip is not nipped between his teeth and the instrument, which emerges from the right side of the mouth (*Fig. 345*). The electric and pneumatic connections are then made and a small amount of air is blown into the stomach.

The first view of the interior with the objective looking downwards (i.e., to the patient's left) is the shallow pool of gastric juice—Henning's 'Schleimsee'—which lies at the most dependent part of the greater curve. The greater curve is recognized at once by the steep longitudinal folds in this region, and as the instrument is rotated forwards and backwards, the anterior and posterior walls are seen. In the normal stomach, moderately inflated, folds are present on the posterior wall, though much less prominent than the steep rugæ on the greater curve itself, but few folds exist on the anterior wall. It is advisable not to attempt at this stage to see the upper part of the lesser curve, as this needs further inflation of the stomach and

is better delayed until the end of the examination. The instrument is now advanced well into the stomach with the objective facing upwards to the patient's right. Unless the stomach is U-shaped, the whole of the pyloric antrum will now come into view (*Fig. 346*), and a little further inflation will stimulate active peristalsis. The waves of contraction can be observed passing from the mid-gastric region steadily towards the pylorus, to end in the relaxation of the sphincter and the pouting of some folds of duodenal mucosa into the orifice, sometimes with the regurgitation of a little yellowish fluid.

The whole of the pyloric region can be examined with ease, and any suspicious area can be particularly closely inspected immediately after the passage of a peristaltic wave when the mucosa of the back of the wave is turned to face the observer. The viscus is then further inflated, although never sufficiently to cause the patient discomfort, and as the instrument is slowly withdrawn the incisura angularis and the lesser curve above it come prominently into view, forming a smooth, rounded arching surface, which gets closer and closer to the objective as it approaches the cardia. The balloon is then inflated, and at once the objective can be seen to stand back from the mucosa, which can now be examined right up to the cardiac orifice.

In examining the greater curve of the stomach, it is often of great help to apply manual pressure to the abdomen, much as is done in radiological examination. The mucosa of this region can by this means be brought closer to the objective for better inspection. Difficulty in seeing the lesser curve of the pyloric antrum can often be countered by asking the patient to take a deep breath; with the cardia fixed, the descent of the liver on the right turns this part of the stomach towards the objective, so that it can be better seen in this way. Again, tipping the table steeply, so that the patient's body is inclined with the head downwards, often relieves the lesser-curve region of the weight of the liver above it in the left lateral position, so that this part of the mucosa springs farther from the objective and can be seen in better perspective. Tipping the table is also useful in moving a collection of bubbles away from an area of mucosa obscured by them. Bubbles are formed by inflating the stomach with the air orifice in contact with the mucosa, and may cause considerable or even insuperable difficulty. For this reason air must be blown into the stomach only when the end of the instrument is in the middle of the cavity and the air aperture (opposite the window of the objective) faces away from the lesser curve and posterior wall.

In examining the stomach endoscopically, notice should be taken of the general size and shape of the viscus, the degree of activity of the peristalsis, and the quantity and viscosity of the mucus. The characters of the mucosa to be noted include the colour, presence of visible blood-vessels, areas of congestion, hæmorrhages or small erosions, and the appearance of the rugæ, whether œdematous, redundant and tortuous, or few and atrophic. Any local lesion may then be described and preferably drawn for future comparison. The examination of the stomach is not complete without a scrutiny of the minute contour of the epithelium, a magnified view of which can be obtained by examining an area of mucosa in very close apposition to the objective of the instrument. The mucosa of the posterior wall of the stomach near the lesser curve is a suitable area for this purpose, and information concerning the nature of diffuse epithelial changes can be obtained in this way.

The examination finished, the gastroscope is gently removed, and the patient is encouraged to bring up the air that has inflated the stomach. Inquiry will reveal that he has undergone no pain, although the pressure on the side of the larynx has usually caused some discomfort. Slight soreness of the throat is not uncommon for one or two days after the examination, but this is seldom severe, and is always relieved by an aspirin gargle or a throat lozenge.

CONTRA-INDICATIONS

The few contra-indications to gastroscopy include those cases where the examination is not possible or justifiable on account of severe illness, especially if associated with dyspnoea. Such cases are beyond the scope of gastric investigation of any kind. Certain local conditions preclude the passage of the gastroscope, such as carcinoma or stricture of the œsophagus or cardiac orifice, gross curvature of the spine, aneurysm of the aorta, intrathoracic neoplasm. Cirrhosis of the liver or congestive heart failure with possible œsophageal varices also contra-indicate the instrumentation. After recent hæmatemesis gastroscopy is inadvisable on account of the risk of air embolism. The examination is also preferably not undertaken during an exacerbation of ulcer pain, as distension of the stomach may possibly cause a perforation of the softened base of an active ulcer. In expert hands, and with only slight inflation of the stomach, the latter two conditions need not absolutely preclude gastroscopy if the circumstances of the case make it a matter of necessity. The one great danger of the older method of gastroscopy—that of perforation of the œsophagus, with fatal mediastinitis—has been completely excluded by the advent of the flexible instrument.

Used with proper regard for contra-indicating circumstances, and with strict adherence to the dictum that no force must ever be used in the manipulation, gastroscopy with the flexible instrument is a method of investigation as completely without danger as the introduction of an ordinary stomach tube.

Henning has performed in his Klinik at Leipzig over 2000 gastroscopies without accident, and other clinicians both abroad and latterly in this country have adopted gastroscopy as a routine method of investigation. Any method of endoscopy, whether of the œsophagus, trachea, stomach, bladder, or rectum, is liable to accident in clumsy or unsympathetic hands. In the diagnosis of disease of the stomach gastroscopy is now to be regarded as corresponding in safety and in usefulness to these other older-established methods of instrumental investigation.

ENDOSCOPIC APPEARANCES OF THE STOMACH

The gastroscopic appearances described in these pages are illustrated by a number of paintings of actual cases made by an artist. The views were sketched in the operating theatre as the cases were demonstrated through the gastroscope.

The Normal Stomach.—The shape of the normal stomach has already been dealt with. In colour the mucosa has the same pink shade as that of the buccal mucous membrane, but is not so pale (*Fig. 346*). No blood-vessels can be seen, and the surface is glassy smooth, while the mucus of the 'Schleimsee' forms a clear limpid pool. The rugæ are clear-cut straight folds, and the troughs between them are quite flat. The height of the folds varies with the degree of distension of the stomach. In the almost empty stomach folds are present over the whole of the surface, except that of

the lesser curve and pyloric antrum. The folds on the anterior wall are few and low, and are soon abolished by slight inflation of the viscus; those on the posterior wall are more marked, but can be abolished by full distension with air. The longitudinal folds marking the greater curve are the most pronounced of all, and can still be seen in the distended viscus.



FIG 346—View of normal stomach looking towards the pylorus.

Gastritis : Hypertrophy : Atrophy.—Chronic inflammatory changes in the mucosa are indicated by general heightening of the colour to a deep red, and at the same time the surface loses its mirror-like smoothness and assumes a matt texture. The pool of mucus is no longer clear, and the secretion hangs on to and between the folds of mucosa in a thick sticky film. Areas of patchy congestion of the mucosa can be seen, and there may be hæmorrhagic extravasations into the sub-mucosa according to the degree of inflammation. The rugæ in uncomplicated gastritis—such as is associated with the presence of a carcinoma—are dull and oedematous, but remain relatively straight.

These are the appearances of *gastritis* alone. It has become habitual to subdivide gastritis into a greater or lesser number of types, and to include, at the least, atrophic, hypertrophic, and catarrhal forms, while Moutier subdivides these into numerous subgroups according to associated manifestations such as hæmorrhages, erosions, etc. As the result of a still small experience of gastroscopy, the author is coming to the conclusion that there is only one form of chronic gastritis—the so-called ‘catarrhal’ type described above. The conditions named ‘atrophic gastritis’ and ‘hypertrophic gastritis’ appear to be combinations of atrophy or hypertrophy of the epithelium with a quite secondary inflammatory change of greater or less degree. Cases have been examined in which there was epithelial hypertrophy or atrophy without any of the signs of inflammation at all, others in which the inflammatory element has been slight, and others in which it has been considerable, while the degree of atrophy or hypertrophy in the various cases has borne no apparent relation to the degree of gastritis.

The appearances of *hypertrophy* of the mucosa are: A bright but uniform red colour of the surface, which is still shiny; close-up inspection shows that the surface is no longer flat but exhibits a very fine smooth velvety nodulation or mammillation, the 'Höckerung' described by Henning, indicative of the epithelial hyperplasia; the mucus in a case without inflammatory changes is clear, but the most obvious characteristic is the redundancy of the rugæ, which are steep and tortuous so that those on the anterior wall are still present when the viscus is moderately distended, while the posterior ones persist in spite of full inflation. These appearances and those of gastritis described above may be superimposed to form the so-called 'hypertrophic gastritis'. It is in this combination of circumstances that multiple small acute erosions commonly occur (Moutier's 'gastritis erosiva'). These are clear-cut, dull-yellow areas, almost level with the general surface of the mucosa. It seems that both hyperplasia (with hyperacidity) and gastritis are necessary before the erosions occur. Whereas in hyperplastic gastritis with or without a chronic ulcer small acute erosions are often seen, these are never seen after a course of treatment when the gastritis has subsided but the hyperplasia remains. In health the resistance of the epithelial cells to digestion by their own secretion appears to afford sufficient protection against even a raised acidity. If, however, the cells are devitalized by inflammatory changes, their defence is impaired and peptic erosions may occur as a result. In the absence of severe gastritis, gastric hyperplasia is invariably associated with a high test-meal acidity. Superimposed inflammation, however, appears to depress the secretory activity of the epithelial cells less than their resistance to auto-digestion, for erosions occur without much diminution of the hyperchlorhydria. Duodenal ulcer (demonstrated radiologically) is the type of erosion most commonly associated with 'hyperplastic gastritis'. The symptoms, however, commonly attributed to the ulcer appear to be mainly referable to the gastric condition, for cases with 'duodenal' symptoms of pain relatively late after meals, relieved by food, bismuth, or alkalis, all have the same characteristic endoscopic appearances, whether an ulcer is demonstrated by the barium meal or not.

The appearances of mucous *atrophy* of the stomach are well seen in pernicious anæmia. In this disease the mucosa is very pale and of a slightly yellowish tinge. The rugæ are ill-defined or absent, the interior of the stomach being a uniform flat surface, rather translucent in appearance, and the branching vessels of the sub-mucosa can be seen through the attenuated superficial layers. The presence of inflammation may be found in the dull œdematous change in the surface and the presence of occasional hæmorrhages, but, again, the degree of atrophy and the degree of inflammation bear no apparent relation to one another.

These observations are entirely in agreement with Hurst's postulation of the congenital hypo- and hyper-sthenic types of stomach which are more prone than the normal viscus to attacks of gastritis, and which in the hypersthenic stomach may give rise to peptic ulceration.

Rare Conditions.—Interesting and unusual conditions may be revealed by gastroscopic examination to elucidate cases of otherwise symptomless hæmatemesis, in which all other investigations are negative. The examination of course is performed some time after the bleeding has ceased. One such case showed a mild degree of diffuse atrophic change with an area of mucosa which was swollen and thickened in bullous fashion, with curious folds unlike the normal gastric rugæ, in that they were short and irregularly arranged. Any possibility of a malignant

condition was excluded by the fact that the folds were soft and flexible and that peristalsis passed normally over this area of the stomach wall. The case was one of a localized area of *polyposis* in an atrophic mucosa. There had been two hæmatemeses separated by a ten-year interval, the second being severe enough to reduce the hæmoglobin to 30 per cent of normal.

Another case of unexplained hæmatemesis, that of an otherwise symptomless young man who had had six severe bleedings in two years, showed a condition of diffuse *gastrostaxis*. The whole of the mucosa was œdematous and dark brownish-red in colour, and was studded all over with submucous hæmorrhages. Blood examination in this patient revealed a very low platelet count which was considered to be the cause of the bleeding, although no hæmorrhages had occurred elsewhere.

Innocent tumours are occasionally seen endoscopically, though they rarely give rise to symptoms. Two examples have occurred in my series, and the endoscopic



FIG. 347.—Case 18. Innocent tumour of stomach, probably a myoma.

picture of one of these is included. *Fig. 347* shows what is probably a myoma. It was discovered accidentally during endoscopic examination of the stomach in a case of severe anæmia. There were no gastric symptoms. The tumour shows the characteristic smooth rounded even contour, and a dilated superficial vein can be seen through the stretched and thinned mucosa covering it. In another case a similar tumour was discovered in the pyloric region. The patient had had occasional attacks of abdominal pain and latterly a regular pain after food. The general characters of the tumour were similar to those of the previous case, but in addition there was an acute peptic erosion of the attenuated epithelium on the summit of the swelling. The importance of these cases lies in their differentiation from carcinoma. In the second case the filling defect was observed radiologically, and the suspicion of carcinoma had been raised and apparently confirmed by the test-meal report of achlorhydria. The true nature of the condition was only discovered on gastroscopy.

Ulcer.—Peptic ulcers occur in a stomach which is already the seat of diffuse hyperplastic and inflammatory changes in the mucosa. All stages of ulceration may

be found between the acute epithelial erosions described above and the typical chronic ulcer. The flat indurated base and clear-cut margins of the latter are well recognized and need no amplification here, but the gastroscope shows in the living subject the contrast between the bright-red mucosa round the ulcer and the yellow colour of the base (*Fig. 348*). Another feature of the gastroscopic view is the ring of œdematous mucosa round the crater of an active ulcer, an appearance which is lacking in autopsy specimens. The appearances of the ulcer vary according to its activity or stage of healing. *Figs. 349–352* show the changes well. They are paintings of the gastroscopic appearances of the same ulcer under medical treatment. *Fig. 349* shows the active ulcer, a deep circular crater with a wide area of smooth œdematous congested mucosa round it. The second picture (*Fig. 350*), taken one month later, shows the changes indicative of healing. The ulcer is now a shallow crater, due to the subsidence of the surrounding œdema, with



FIG. 348.—*Case 51.* Chronic peptic ulcer on lesser curve (healing). Note: clear-cut edges of yellow base; radiating cicatrices in surrounding mucosa; 'hitched-up' appearance of incisura angularis.

newly-formed epithelium encroaching from the edge, which is no longer circular and undermined but crenated and shelving down to the level of the ulcer base. It will be noted that the red granulating surface typical of a healing ulcer elsewhere does not occur with peptic ulceration. The most striking change, however, lies in the radiating folds of mucosa surrounding the ulcer. These are due to submucous cicatrization in the region of the crater which has taken the place of the inflammatory elements. In the third picture, three weeks later still (*Fig. 351*), the stellate area of mucosa is now seen to be projecting somewhat into the cavity of the stomach. This is due to the contraction of the scar tissue in the wall of the stomach under the base of the ulcer, as well as in the surrounding submucosa, so that the ulcer and surrounding mucosa are bunched up and invaginated into the lumen. This is an indication that the ulcer is not adherent in the lesser sac—a point of prognostic importance. The fourth picture, three months later (*Fig. 352*), shows the ulcer completely healed. The scar tissue is fully contracted and the mucosa has evened itself out again over the surface.

The apparent size of an ulcer seen endoscopically varies with the distance from the objective of the instrument. Thus one must not be misled by the apparent small size of pyloric ulcers compared with the ulcer high up on the lesser curve.



FIG 349—*Case 12* Atrophic chronic peptic ulcer, untreated. Note surrounding inflamed mucosa—œdematous, but with no radiating folds indicative of healing.

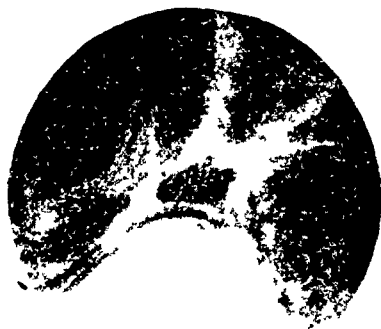


FIG 350—*Case 12* Same ulcer after 4 weeks' medical treatment. Note signs of healing: radiating cicatrices, shallow crater, crenated border.



FIG 351—*Case 12*. Same ulcer after 7 weeks' treatment. Note elevation of area of ulceration into the lumen (*see text*), crater almost disappeared.



FIG 352—*Case 12* Same ulcer healed. View taken three months after discharge from hospital. Note mucosa of region of old ulcer has become level again—the stellate scar is almost abolished.

With some experience it is often possible to estimate the size of an ulcer by its relation to the size of the bow of the lesser curve or the diameter of the pyloric antrum. More important and easier is to report on the increase or decrease in size of any given ulcer on a second inspection after a course of therapy.

The degree and extent of cicatrization of the stomach wall near an ulcer can be observed gastroscopically, and the presence of adhesions in the lesser sac can be judged from the 'hitched up' appearance of the ulcer and its fixity in spite of



FIG. 353.—Case 37. Acute gastro-jejunal ulcer. Note: pylorus; valvulae conniventes of jejunum; acute peptic erosion almost level with gastric mucosa.



FIG. 354.—Case 60. Chronic gastrojejunal ulcer. Note: acute extension down the jejunum; excrecence of grossly inflamed gastric mucosa overhanging ulcer crater; efferent loop of jejunum; pyloric antrum (on left).



FIG. 355.—Case 4. Part of the edge of a gastro-jejunal stoma with underlying chronic anastomotic ulcer. Note: efferent loop of jejunum with valvulae conniventes, polypoid excrecence of grossly inflamed gastric mucosa (the ulcer lay beyond this).

manipulation of the abdomen or tilting the patient. At a second inspection, if, although the ulcer is healing, it is not level with the gastric wall or even invaginated a little as described above, then its adherence in the lesser sac is confirmed. These points are of value in giving a prognosis of the prospects of cure by medical means.

Ulcers on the anterior or posterior walls tend to be hidden by the redundant folds of hypertrophied mucous membrane, and are less obvious than the lesser-curve ulcers, which make a striking contrast to the normal smooth contour of this region. In a J- or U-shaped stomach an ulcer of the lesser curve of the pyloric antrum cannot be directly seen, but in such cases there is a peculiar and characteristic notching of the normally rounded contour of the incisura angularis. This appearance has often led the author to suspect the presence of such an ulcer, which after some manipulation and tilting of the patient has at length been brought into view.

Anastomotic Ulcer.—Endoscopic examination of a stomach which has been operated upon, usually for the performance of gastro-enterostomy, presents special difficulties on account of the distortion of the stomach which may be present, but chiefly because the viscus can seldom be satisfactorily distended with air, which tends to escape through the stoma. Moreover, the presence of a double loop of gut between the stomach and the already prominent vertebral column tends still further to lift the posterior gastric wall forward, thus turning the stoma away from the direction of the instrument, which lies in the paravertebral depression in front of the left kidney. It was on this account that the balloon was designed, and this has been found to be of great help in obtaining a view of the stoma. The opening itself is instantly recognizable (*Figs. 353-355*), for the transverse valvulæ conniventes of the jejunum seen through the stoma are unmistakable. The instrument can often be made to pass for some inches along the efferent loop of intestine, and with the aid of the balloon the jejunal mucosa can be inspected. In spite of the difficulties it is possible to obtain a satisfactory view of the stoma in the great majority of cases. It must be admitted, however, that in the author's experience stomata without ulceration are much easier to visualize than those with an ulcer. It is therefore much easier to establish the absence of a gastro-jejunal ulcer when suspected than directly to observe its presence. The reasons for this lie in the extra distortion of the stomach and the stoma by the ulcer, and also in the intense gastritis that occurs near the ulcer, which becomes hidden from view by the overhanging œdematous folds of hypertrophied mucosa. This latter handicap can nevertheless be turned to good account in diagnosis, for a localized patch of intense gastritis on one side of the stoma, in contrast with a lesser degree of gastritis elsewhere, is strong evidence of the presence of an ulcer, the crater of which may be hidden from the objective of the instrument (*Figs. 354, 355*).

'Post-operative' Gastritis.—Although the great majority of cases *with symptoms* after gastro-enterostomy have a greater or less degree of gastritis, I cannot subscribe to the view of some authors that gastritis is the invariable result of the performance of gastro-enterostomy. In spite of the fact that all my cases have had symptoms which have been the indication for the gastroscopy, I have seen three uninfamed stomata. In each the gastro-jejunal junction was a thin line where the two types of mucosa were in apposition. The gastric and jejunal surfaces were of uniform colour and possessed the normal highly reflecting surface, although the mucosa of the stomach showed the tortuous redundant rugæ indicative of hyperplasia.

In the interpretation of post-operative gastroscopic appearances, it is well to remember that the operation was performed for peptic ulceration complicating chronic gastritis in a stomach with a hyperplastic mucosa. Even under medical

treatment of such a condition the hyperplasia persists though the inflammation may subside, and it is therefore not surprising that after gastro-enterostomy the stomach still exhibits a hyperplastic mucosa which may again become the seat of



FIG. 356.—Case 53. Early carcinomatous ulcer on anterior wall of stomach. Note: infiltration of surrounding submucosa, which is raised into low mounds by the growth beneath.



FIG. 357.—Case 7. Ulcerating 'massive' carcinoma of greater curve. Note: raised everted edge of growth; sloughing base of ulcer.



FIG. 358.—Case 24. Upper limit of carcinoma of lesser curve. Note: 'rigid' appearance; submucous hemorrhages; nodules of growth in submucosa.



FIG. 359.—Case 23. Carcinomatous ulcer of lesser curve. Note: surrounding infiltration of mucosa; nodular sloughing base.

a recurrent or chronic inflammatory change. It is indeed not claimed for gastro-enterostomy that it cures or prevents the recurrence of the gastritis which is the underlying cause of the peptic ulcer, but that it reduces the gastric acidity to a

point at which the devitalized epithelial cells can resist its eroding action. The operation is only to be considered as removing the dangerous and troublesome complication of ulceration from the otherwise relatively harmless disorder of hyperplastic gastritis. But to ascribe the antecedent gastritis to the operation is hardly in accordance with the pathological sequence of events.

Carcinoma.—The superficial appearance of carcinomata of the stomach also needs no description here, except in regard to the special additional points which can be observed gastroscopically, by virtue of the fact that the growth is alive and the blood circulation is going on within it (*Figs. 356-359*). Thus the massive carcinoma, with or without central necrosis and ulceration, is of a high colour, and hæmorrhages may be seen within it. Occasionally pale submucous nodules of growth can be seen in contrast with the red gastric mucosa near the tumour, an appearance not observed in colourless autopsy specimens. One can observe the rigid character given to the stomach wall by the malignant infiltration in contrast to the soft flexible surface elsewhere, which alters in shape with the degree of distension and with respiratory and cardiac movements.

The gastroscope is unfortunately of little use in assessing the operability of a growth. Not only are metastases in the liver or lymph-nodes beyond its field, but the area of stomach wall above a visible growth cannot be pronounced free from infiltration. Only the mucosal aspect is visible, and the spread of carcinoma in the subperitoneal lymph-plexus of the stomach cannot be detected from within.

Gastroscopy is contra-indicated with a growth at the cardia. For this reason alone X-ray examination is a necessary preliminary to gastroscopy if this condition is suspected.

THE CLINICAL VALUE OF GASTROSCOPY

It is obvious that direct inspection of the living gastric mucous membrane is scientifically a matter of the first importance for that development of our knowledge of the pathology of the stomach which alone can lead to improved methods of clinical therapy. The object of this paper, however, is to show that gastroscopy has a useful place in the investigation of ordinary clinical cases notwithstanding the invaluable help of modern radiological technique. Further, that in many cases gastroscopic examination can only be omitted at the expense of the patient, either by submitting him to unnecessary laparotomy or to inadequate treatment through lack of a sufficiently accurate diagnosis. It must be emphasized, however, that gastroscopy is the last investigation that should be made upon a patient, and that, for reasons which will subsequently be discussed, it should not be performed without previous radiography of the stomach.

Carcinoma.—If in the investigation of a case, after consideration of the clinical evidence, the radiological report, and the results of test-meal and occult-blood examinations, a diagnosis is definitely established upon which operation is indicated, such as carcinoma with a gross filling defect, then gastroscopy is not required. In a number of cases, however, there is an element of doubt in the diagnosis largely due to the circumstantial nature of the evidence, except that of the radiograph. The interpretation of the test-meal and occult-blood examinations is notoriously problematical, and in the doubtful case the onus of diagnosis is therefore thrown upon the radiologist. In these circumstances the important

decision whether to operate or not often turns on the radiologist's opinion of a suspicious area of the stomach, about which he cannot be certain. In the past it has been a not uncommon occurrence for a patient, suffering from no more than an atrophic gastritis, but under suspicion of carcinoma, to be submitted to laparotomy, on the plea that it is better to operate and to be mistaken than to miss a growth while it is still operable. Gastroscopy now provides the means whereby such mistakes can be averted, and is most strongly indicated in cases of doubtful carcinoma where the radiologist cannot give absolute confirmation of the diagnosis. If the doubtful area of the stomach suspected by the radiologist can be brought before the objective of the gastroscope, and the gastroscopist can say whether this has been done or not, then a definite answer can be given to the question—is there or is there not a carcinoma present? In this way a diagnosis of carcinoma can be established in the early, still doubtful case. It is generally accepted that the chief factor in any improvement that may be achieved in the treatment of malignant disease, especially of the stomach, is early diagnosis, and it is in this field that gastroscopy is so valuable an addition to the available methods of investigation.

In the case with symptoms referable to the stomach and a completely negative radiograph, gastroscopy is nevertheless still indicated. Apart from the desirability of establishing the degree and type of gastritis in such circumstances, cases do occur, although rarely, in which an early carcinoma or a posterior-wall ulcer has been overlooked on radiological examination. The records of any hospital will show cases which, having been admitted for gastric symptoms and discharged as 'gastritis' after negative results from X rays and test-meal, have been re-admitted some time later with advanced carcinoma of the stomach. Such a case came to the post-mortem room recently in which a carcinoma of the pylorus had been undiagnosed at a previous investigation because the presence of a gastro-enterostomy opening made years previously had diverted the barium meal and prevented the radiologist from outlining the growth. Gastroscopic examination at the time of investigation would probably have revealed an early carcinoma which might have been successfully removed.

Gastritis.—If, happily, a carcinoma be not found on gastroscopy and the radiological report be confirmed in this respect, great importance is still to be attached to the identification of the type of generalized disease of the mucosa which is giving rise to the symptoms. The view that peptic ulceration is a sequel of an untreated 'acid' gastritis in a hyperplastic stomach is rapidly commanding acceptance. An accurate diagnosis of this condition before the stage of ulceration, only possible by means of the gastroscope, will enable prophylactic measures to be taken against the supervention of ulceration. Alternatively, in the hypoplastic type of stomach, gastritis is now established as a primary etiological factor in the production of certain forms of anæmia, so that attention to the stomach is of the first importance in the treatment of, or prophylaxis against, this type of disease. Also, the view is gaining ground that carcinoma of the stomach, as with carcinoma elsewhere, may be a possible consequence of the chronic irritation of gastritis in an atrophic mucosa, although this is as yet non-proven. The gastroscope has done much towards the restoration of 'gastritis' as a definite pathological entity. It now appears that this much neglected clinical condition may be fraught with serious consequences, so that in its diagnosis and treatment gastroscopy may prove to be a major influence in the reduction of the incidence of its better recognized and more dangerous sequelæ.

Ulcer.—As with carcinoma, if a gastric ulcer is found on X-ray examination of a case in circumstances which indicate operative treatment, again gastroscopy is not required. But if doubt as to the advisability of operative treatment exists, having regard to the poor general condition of the patient or to the possibility of cure by medical means, then gastroscopy can obtain the evidence whereby the exact nature of the ulcer can be established and the correct course of treatment determined. The gastroscopic appearances of the acute erosion, the chronic ulcer, or the healing ulcer are for the most part well-defined and easily recognized, and a confident opinion on the case can usually be given by the gastroscopist.

The establishment of an exact diagnosis is not, however, the limit of usefulness of the gastroscope in the field of peptic ulceration. If an ulcer is to be treated medically, then the gastroscopist can inform the physician of the effect of his treatment both on the ulcer and on the associated gastritis. It is often a very difficult matter to know when a gastric ulcer is healed and the patient can be allowed to return to normal life. X rays do not really demonstrate an ulcer—they outline a crater formed, not by the ulcer, but by the œdematous mucosa on either side of it. This œdema subsides rapidly under treatment, and the barium meal may fail to show a crater long before healing is complete, particularly if the ulcer lies to one or other side of the lesser curve. An instructive case of lesser-curve ulcer occurred recently, which was considered healed after five months' medical treatment. Repeated radiographs had shown the crater to have disappeared, yet the ulcer perforated a fortnight after the patient had been discharged from hospital! Gastroscopy, by rendering the ulcer open to direct inspection, now enables the stages of healing to be observed and the completion of the process to be confidently determined (*see Figs. 349-352*). If, on the other hand, the ulcer is not responding to treatment, then gastroscopy will establish this fact beyond question, and surgical measures can be undertaken without loss of time in useless therapy.

The supervention of a malignant change in a chronic ulcer is not always visible to the naked eye, and therefore cannot always be diagnosed at a first gastroscopic examination. Where, however, considerations of grave operative risk justify a delay in radical treatment of a doubtful case, a second inspection of the ulcer after a month's medical treatment will reveal whether it is healing and therefore innocent, or progressing and therefore probably malignant. The value of this method is exemplified in the type of necrotic ulcer occurring in the atrophic senile stomach, which often closely resembles a carcinoma in the shortness of the history, the loss of appetite, loss of weight, the associated achlorhydria, and the large size of the ulcer. These cases, however, readily respond to medical therapy, so that the decision as to malignancy or innocence need not be delayed for longer than a month. In these old patients such a delay in operative treatment of the malignant case is more than compensated by the satisfactory healing of the ulcer in other cases, where an extensive operation might have led to a fatal issue.

The value of gastroscopy in the assessment of new methods of therapy such as the recently introduced histidine treatment is evident, but it is beyond the scope of this paper to enlarge on this aspect of the method.

Gastro-jejunal Ulcer.—In the diagnosis of gastro-jejunal ulceration gastroscopy is an investigation of special value. The presence of the stoma greatly diminishes the value of the test-meal, and it is well known that anastomotic ulcers are

notoriously difficult to show radiographically by the barium meal. The reports of *Cases 4 and 60* are a striking illustration of this. In each of these, X-ray examination had produced negative results on more than one previous occasion; and even immediately preceding the observation of the ulcer through the gastroscope, the screen had failed to find evidence of any pathological change in the stoma. Moreover, in this type of case the clinical picture of gastro-jejunitis without ulceration is so similar to that of an actual anastomotic ulcer that clinical diagnosis is a matter of extreme difficulty. In such a case, if the stoma can be brought into the field of the gastroscope a definite observation can be made as to the presence or absence of a gastro-jejunal ulcer. It has been pointed out that the gastro-enterostomized stomach presents special difficulties to the gastroscopist, but in spite of this a definite diagnosis can be made in the great majority of cases.

RELATION OF GASTROSCOPY TO RADIOLOGY OF THE STOMACH

The advent of gastroscopy does not in any way detract from the value of radiology in gastric investigation. The barium meal often suffices alone to establish the diagnosis, and must always be the routine method of examination of the stomach. Moreover, gastroscopy, to be used safely and to the best advantage, will always be to a certain extent dependent on previous radiology, the two methods of investigation being complementary to each other in their results. X rays demonstrate the size, shape, position, and movements of the stomach, and reveal gross filling defects or deep ulcers. The gastroscope can extend this description to include the more minute characters of the mucosa—the presence of inflammation, hyperplasia, atrophy, hæmorrhages, or erosions; or the radiological silhouette of a suspicious area can be amplified by a description of the colour, texture, and surface configuration of the lesion. In this way radiology and gastroscopy combine together to provide a complete and accurate description of the stomach, so that, in the words of Henning, an exact morphological diagnosis can now be established in every case of gastric disease.

STATISTICAL ANALYSIS OF CASES

The first sixty cases gastroscopied at the London Hospital up to June, 1936, have been summarized and included as a table to illustrate the general use of gastroscopy (*see pp. 494–500*). The examination was not done as a routine, but only in cases where there was a definite indication for its employment. It will be seen that ten cases fall into the category in which there has been suspicion of carcinoma, either supported or not excluded radiologically, but in which no carcinoma has in fact been present. In most of these cases gastroscopy was a decisive factor in the avoidance of an exploratory operation. In three of them a suspicious ulcer was visible on the screen, but its innocent nature was established by the gastroscope either at once or after a short course of therapy. On the other hand four cases of dubious carcinoma were confirmed on direct inspection.

Three patients with symptoms of ulcer and supporting radiological evidence such as ‘spasm’ were found to be cases of gastritis only.

Two of these ‘negative’ cases had actually, in the past, had a fruitless laparotomy, so suggestive were the symptoms, while in two more operation was performed

in spite of contra-indicative gastroscopic findings, in each case with negative results (*Cases 6 and 51*).

Sixteen cases of doubtful gastro-jejunal ulcer were examined; the stoma was visualized in fourteen of these, and in eight cases anastomotic ulcer was definitely excluded. In six cases (*Cases 4, 8, 10, 22, 37, and 60*) an active ulcer was found, on three occasions by direct inspection, and in the other three cases by deduction from the presence of a localized polypoid excrescence of highly inflamed gastric mucosa at one part of the stoma. Of the three ulcers directly observed, two were fairly large acute erosions of the mucosa on the gastric side of the stoma, and one was a deep chronic ulcer. The three ulcers inferred from the polypoid excrescence of gastric mucosa over them were *ipso facto* chronic ulcers. In all, therefore, there were four cases of chronic gastro-jejunal ulcer, of which only one was directly visible. The cause of the symptoms in the sixteen unsatisfactory cases after gastro-enterostomy therefore was: Eight cases—persistent severe hyperplastic gastritis. Two cases—hyperplastic gastritis with acute gastro-jejunal erosion. Four cases—chronic anastomotic ulcer, also with gastritis. Two cases—normal gastric mucosa; normal stoma; symptoms not due to stomach.

A lesion in an unusual situation (other than gastro-jejunal ulcer) has been discovered in four cases (*Cases 9, 21, 53, and 59*) where the condition was not apparent on X-ray examination. Three of these were peptic ulcers on the posterior wall and one was an early carcinomatous ulcer on the anterior wall (*Case 53*). In two cases the presence of an ulcer which was doubtful radiologically has been confirmed.

SELECTED CASE REPORTS

Although the tabulated list of cases on pp. 494–500 gives some indication of the clinical value of the gastroscope, perhaps the following brief notes of some of the more interesting cases in the series provide a better demonstration of its use.

Case 4.—J. L. G., male, aged 53. Gastro-enterostomy in 1923 for duodenal ulcer. Symptoms have persisted since operation. Re-admitted in 1934 for investigation—? gastro-jejunal ulcer, with negative results from X rays and test-meal. Symptoms progressed. Admitted again in 1935 for further investigation, in poor condition. Again test-meal showed absent HCl and X rays showed a “patent freely-working stoma” with no evidence of ulceration. Gastroscopy showed a localized polypoid excrescence of highly inflamed mucosa projecting from the gastro-jejunal junction (*see Fig. 355*). The deduction was made that a gastro-jejunal ulcer was present on the jejunal side of the stoma beyond this area. At operation by Sir James Walton a large adherent gastro-jejunal ulcer was found and partial gastrectomy was performed. Examination of the specimen showed the ulcer rather on the jejunal side of the stoma under cover of the oedematous mucosal projection that had been observed gastroscopically.

Case 6.—E. S., female, aged 46. Long history of indigestion, clinically due to viscerop-tosis. Test-meal showed 0.18 per cent free HCl. X rays showed evidence of duodenal ulcer. Gastroscopy showed a moderate degree of gastritis in a rather atrophic mucosa. The opinion was given that duodenal ulcer was very unlikely in the absence of hyperplastic gastritis. Operation, however, was done as the X-ray findings were definite. No ulcer was discovered.

Case 10.—L. D., male, aged 48. Gastro-enterostomy eight years previously. Had been symptomless since operation until admitted with acute onset of diarrhoea, foul flatulence per mouth, vomiting of faeculent material, and rapid wasting. At operation a gastro-jejuno-colic fistula at the site of ulcer was found, but the condition of the patient prohibited anything further than the separation of the colon from the anastomosis. Patient did well after operation and had no symptoms as before, but gastroscopy eleven months later showed evidence of

another 'silent' gastro-jejunal ulcer. Strict medical treatment was commenced. Ten months later the scar of a healed gastro-jejunal ulcer was observed. Treatment relaxed.

Case 20.—J. W., male, aged 53. Twenty-eight years' history of indigestion, latterly increasing in severity. Test-meal showed 0.31 per cent free HCl, but the radiologist found a "very unusual appearance" of the stomach on which he was unwilling to give an opinion. The case was considered to be one of visceroptosis, but gastroscopy revealed the pylorus apparently situated in the middle of the lesser curve, adjacent to an ulcer. At operation this was found to be indeed the case. An old-standing large lesser-curve ulcer in a U-shaped stomach had resulted in the adhesion together of the limbs of the U, with subsequent erosion and disappearance of the resulting septum between the two halves of the stomach. The pylorus was therefore left opening into the stomach about three inches from the cardia. Partial gastrectomy was performed. Did well.

Case 21.—A. C., male, aged 63. Gastro-enterostomy 6 months ago for high adherent lesser-curve ulcer. Symptoms of ulcer persisted, but X rays showed no evidence of it. Gastroscopy showed the ulcer high up near the cardia, smaller, but still unhealed. Patient was put on a stricter course of treatment, and a second gastroscopy 5 weeks later showed the ulcer to be healed. Treatment was relaxed and the patient has kept well since.

Case 26.—F. G., male, aged 51. Short history of indigestion. Test-meal showed raised acidity. X rays showed small lesser-curve ulcer. Patient admitted as suitable for trial of histidine therapy. Fourteen days later, before treatment began, patient was gastroscopied, but ulcer was found to have already almost healed in the interval.

Case 32.—R. L., male, aged 52. Fifteen years' vague history suggestive of visceroptosis. X rays showed a dubious area on the lesser curve, but this was discounted by the clinician, who considered the symptoms to be due to the visceroptosis. Gastroscopy showed an adherent lesser-curve ulcer. After one month's treatment a second inspection showed no evidence of healing. Gastrectomy was therefore performed.

Case 37.—H. H., male, aged 43. Had been well since gastro-enterostomy 9 years previously for duodenal ulcer until a recent attack of epigastric pain and vomiting. X rays showed a normal stoma but an abnormal pylorus suggestive of carcinoma. Gastroscopy showed this appearance to be due to extra-gastric adhesions, but a large acute peptic erosion was observed at the stoma (*see Fig. 353*). The patient was put on strict medical treatment and has remained well since.

Case 51.—W. J., male, aged 44. Short history of indigestion, vomiting, and some loss of weight. Test-meal showed diminished secretion and X rays showed an ulcer on the lesser curve. The physician and surgeon considered the case to be a malignant ulcer. Gastroscopy showed the ulcer with all the signs of healing (*see Fig. 348*). The other aspects of the case were felt to be so suggestive that operation was performed two weeks later. No evidence of carcinoma was found. Wedge resection and gastro-enterostomy was performed. The excised ulcer was found to be half covered with newly formed epithelium.

Case 52.—M. C., male, aged 64. Ten years' history suggestive of ulcer, with a recent change in symptoms to include vomiting, loss of weight, and severer pain. Test-meal showed complete achlorhydria and X rays showed an "uneven lesser curve—? carcinoma". Gastroscopy showed a lesser-curve ulcer with signs of healing, although a clinical diagnosis of carcinoma was considered probable in spite of the endoscopic appearance. In view of *Case 51* and the fact that the patient was a very bad risk for operation, a course of medical treatment was given. A second gastroscopy 6 weeks later showed active healing of the ulcer. Carcinoma was therefore definitely excluded and operation avoided. Three months later a third gastroscopy showed the ulcer reduced to a tiny speck—just on the point of complete healing.

Case 53.—J. B., aged 48. Two years' vague indigestion. No abnormal physical signs: X-ray and test-meal normal. Gastroscopy revealed an atypical ulcer on the anterior wall adjacent to a rounded elevation of mucosa (*see Fig. 356*). The suspicion of malignancy raised was discounted for lack of supporting evidence in test-meal or X-ray or clinically. Patient discharged himself after three weeks' medical treatment and did not

return for five months—again with vague epigastric discomfort. Gastroscopy now showed a large sloughing ulcer in a mass of growth. Operation by Mr. Alan Perry revealed an inoperable carcinoma of the greater curve and fundus, adherent to the pancreas.

The interest of this unsatisfactory case is that the malignant character of the ulcer was really observed very early, but through lack of experience was not recognized. One is led to hope and expect that such an error would not be repeated, and that gastroscopy will in future be able to diagnose a carcinoma before any other method of investigation while the symptoms are slight.

Case 57.—C. P., male, aged 52. Gastro-enterostomy three years previously. Operated upon three months ago for perforation of a gastro-jejunal ulcer on the efferent loop of the jejunum, which was sutured. On gastroscopy the whole of the efferent loop of gut was clearly seen, but there was no trace of ulcer. The ulcer therefore had been an acute one and had completely healed after the suturing.

Case 60.—S. S., male, aged 56. Gastro-enterostomy for duodenal ulcer in 1933. Indigestion persisted after operation. Investigation by X rays and test-meal in 1934 showed no evidence of anastomotic ulcer. Re-investigation in 1935 produced the same negative result. In 1936 patient admitted for gastroscopy. Test-meal showed 0.15 per cent free HCl. X rays once more showed no evidence of a gastro-jejunal lesion. Gastroscopy revealed a deep chronic ulcer crater on the gastro-jejunal junction opposite the efferent loop of intestine (see Fig. 354). There was in addition an acute extension of the ulcer down the jejunum, while the gastric mucosa overhung the crater in the characteristic polypoid excrescence. The findings were confirmed at operation by Sir James Walton, and partial gastrectomy was successfully performed.

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY
PERFORMED UP TO JUNE, 1936

CASE NO.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE : ENDOSCOPIC APPEARANCES
1	Distortion by band	0	'Spasm' of antrum	History very suggestive of ulcer, including hæmatemesis and melæna. X-ray supported the possibility. Operation avoided through gastroscopy N.B.—A previous laparotomy had been performed years ago for the same symptoms and no lesion found
2	Hyperplastic mucosa	0.26	Ptosis; no ulcer	History suggestive of ulcer. This excluded. X-ray confirmed. N.B.—Presence of hyperplasia without gastritis
3	'Catarrhal' gastritis	0	No abnormality	History suggestive of carcinoma. Test-meal suggestive. Recent hæmatemesis. Carcinoma excluded by gastroscopy. X-ray confirmed
4	Gastro-jejunal ulcer	0	Normal stoma; no ulcer seen	Gastro-jejunal ulcer diagnosed by gastroscopy (Fig. 355) (<i>case report in text</i>) in spite of negative test-meal and X-ray. Partial gastrectomy
5	Normal stomach	0	—	Case of unexplained vomiting; ? mesenteric adenitis. Gastroscopy excluded gastritis

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE : ENDOSCOPIC APPEARANCES
6	Atrophic gastritis	0.18	Duodenal ulcer ; ptosis	Gastroscopic opinion given that duodenal ulcer was very unlikely with atrophic mucosa. Operation performed. No ulcer found
7	Carcinoma	—	Carcinoma (some doubt)	Carcinoma observed. X-ray confirmed. Partial gastrectomy. (<i>See Fig. 357</i>)
8	Acute gastro-jejunal ulcer ; gastritis	—	—	Clinical diagnosis of chronic gastro-jejunal ulcer excluded. Acute anastomotic ulcer seen. Patient put on strict medical treatment. Now much improved
9	Posterior-wall ulcer	—	No abnormality	Ulcer discovered on posterior wall on gastroscopy. Ulcer had failed to show radiologically on account of its position. Medical treatment instituted. Second gastroscopy : showed ulcer still present. Feeble patient. Treatment continued
10	Gastro-jejunal ulcer	—	—	Patient had had a gastro-jejuno-colic fistula as first symptom of a 'silent' gastro-jejunal ulcer. Colon separated off at operation—ulcer not then active. Gastroscopy suggestive of recurrence of gastro-jejunal ulcer—again without symptoms. Medical treatment commenced. Second gastroscopy 10 months later showed the scar of a healed anastomotic ulcer. (<i>Case report in text</i>)
11	Hyperplastic gastritis	0.18	No abnormality	History suggestive of ulcer. Negative X-ray confirmed. Treatment given for gastritis, with improvement
12	Lesser-curve ulcer	0.20	Showed ulcer crater	Gastroscopic control of medical treatment. Stages of healing observed (<i>see text</i>). Completion of healing determined. Treatment relaxed. (<i>See Figs. 349-352</i>)
13	Carcinoma	0	? Carcinoma (some doubt)	Gastroscope held up just within cardia by growth. Nil seen. Instrument used only as a sound. Operation : inoperable carcinoma
14	Hypertrophic gastritis	0.20	Spasm of pylorus ; ? pyloric ulcer	Pyloric ulcer excluded (duodenal ulcer still possible). Treatment given for gastritis

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE : ENDOSCOPIC APPEARANCES
15	Duodenal ulcer	0.15	? Juxta - pyloric ulcer	Pyloric ulcer excluded. Much spasm of antrum observed. Duodenal ulcer suggested. Course of histidine therapy. Second gastroscopy showed no improvement. Operation performed, adherent duodenal ulcer found. Gastro-enterostomy
16	Non - pathological gastro - enterostomy stoma	—	No abnormality	Hyperplastic mucosa. No gastritis. Whole stoma observed : ulcer excluded. Gastro-jejunal junction a thin, clearly marked line
17	Pernicious anæmia	0	No abnormality	Carcinoma suspected by blood in test-meal. This excluded by gastroscopy. U-shaped stomach. Mucosal atrophy noted.
18	Benign tumour ; atrophic gastritis ; anæmia	0	No abnormality	Carcinoma suspected. No response to treatment for anæmia. Malignant growth excluded by gastroscopy (<i>see Fig. 347</i>)
19	Carcinoma	0	Carcinoma	Gastroscopy to determine operability. Growth reported operable, as normal mucosa above it was sufficient for gastrectomy. At operation, subperitoneal lymphatic infiltration of whole stomach—inoperable <i>N.B.</i> —Gastroscopy useless to determine operability
20	Lesser-curve ulcer ; gross distortion	0.31	Appearances inexplicable ; ? carcinoma	Long vague history. Case considered clinically to be visceroposis. Gastroscopy showed the ulcer and distortion. Partial gastrectomy
21	High posterior-wall ulcer ; stitch ulcer near gastro-enterostomy	0	No ulcer seen ; normal stoma	Ulcer observed. Also bunch of granulations near stoma — ? round a stitch. Medical treatment. Second gastroscopy showed ulcer healed. Patient discharged
22	? Gastro-jejunal ulcer	0.13	Normal stoma	Indirect evidence of gastro-jejunal ulcer—a localized area of gastritis at one part of the stoma. Medical treatment
23	Carcinomatous ulcer	0	Ulcer ; ? carcinoma (some doubt)	Patient in poor health. Carcinomatous nature of ulcer confirmed. Medical treatment otherwise intended. Partial gastrectomy. (<i>See Fig. 359</i>)

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE : ENDOSCOPIC APPEARANCES
24	Carcinoma	0	Carcinoma	Gastroscopy showed growth operable as regards mucosa above it. Laparotomy—extensive involvement of pancreas. Inoperable. (See Fig. 358)
25	Lesser-curve ulcer	0.11	Ulcer seen ; ? malignant	Short history suggestive of carcinoma. Ulcer seen to be a typical peptic ulcer already healing. Operation nevertheless performed on other evidence. Non-malignant ulcer found. Cautery excision and gastro-enterostomy
26	Lesser-curve ulcer	0.12	Showed ulcer	Gastroscopy performed to observe effect of histidine therapy. Ulcer found to be almost healed before treatment commenced!
27	Distortion of stomach ; ? cause	0	? Lesser-curve ulcer	First X-ray examination suggested ulcer. Repeat X-ray did not confirm this. Gastroscope held up on posterior wall by obstruction 2 in. within cardia. Gastritis. Obstructing agent not visible. No operation.
28	Gastro-jejunitis	—	No abnormality	Case suggestive of gastro-jejunal ulcer. Stoma well seen ; ulcer excluded. Medical treatment
29	Hyperplastic gastritis ; jejunitis	0	Normal stoma	Case suggestive of gastro-jejunal ulcer. Stoma well seen ; ulcer excluded. Medical treatment. N.B.—Gastritis much more intense than jejunitis
30	Hyperplastic gastritis ; distorted stoma	—	—	Case suggestive of gastro-jejunal ulcer. Abnormal funnel-shaped stoma seen as though pulled upon. Ulcer excluded
31	Distortion of stomach ; normal stoma	0	? Pyloric lesion ; inefficient stoma	Dyspepsia after gastro-enterostomy. Stoma well seen. No inflammation. Pyloric lesion excluded. Ridge observed due to band across lesser curve
32	Lesser-curve ulcer	0.15	Dubious ; ? ulcer present	Ulcer demonstrated. Medical treatment. Second gastroscopy showed no improvement. Ulcer seen to be adherent. Partial gastrectomy
33	Gastro-jejunitis	0.1	No abnormality	Whole stoma seen. Mild inflammatory changes. Gastro-jejunal ulcer excluded

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1939—*contd.*

CASE NO.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE : ENDOSCOPIC APPEARANCES
34	Polyposis	0.1	No abnormality	A case of unexplained recurrent hæmatemesis (twice in ten years). Polyposis observed
35	Normal stomach ; carcinoma of liver	0.09	Filling defects ; ? carcinoma of stomach	Filling defects observed to be the result of pressure from without (liver). Operation—carcinoma of liver
36	Carcinoma	0	Dubious ; ? carcinoma high up	Upper end of growth observed. Inoperable
37	Gastro-jejunal ulcer	0.1	Normal stoma ; ? carcinoma of pylorus	Acute erosion seen on gastro-jejunal junction. Pylorus observed : carcinoma excluded. Medical treatment. (<i>See Fig. 353</i>)
38	Gastro-jejunitis	0	Tenderness over stoma ; no ulcer seen	Case suggestive of gastro-jejunal ulcer. Whole stoma seen. Ulcer excluded. Medical treatment
39	Benign tumour ; acute ulcer	0	Filling defect	Feeble patient. Case suggestive of carcinoma. This excluded. Acute ulcer seen on top of benign tumour. Operation avoided. Medical treatment
40	Hyperplastic gastritis	0	Normal stoma ; ? carcinoma of pylorus	Gastritis observed. Failed to see into stoma, which was turned away from instrument. Pylorus seen. Carcinoma excluded. Medical treatment
41	Gastro-jejunitis	0.04	—	Whole stoma seen. Ulcer excluded. Marked hyperplastic gastritis ; Mild jejunitis
42	Atrophic gastritis	0	Dubious ; ? carcinoma of pylorus	Whole stomach seen. Carcinoma excluded
43	Lesser-curve ulcer	0.07	Ulcer demonstrated	Failed to see ulcer owing to distortion of stomach obstructing instrument. Another healed ulcer observed high up
44	Normal stomach	0.1	No abnormality	Case of unexplained abdominal pain and hæmatemesis. Whole stomach seen. Gastric lesion excluded
45	Lesser-curve ulcer	0.19	Carcinoma of lesser curve	Typical peptic ulcer observed. Second gastroscopy after one month showed little healing ; much scarring ; ulcer adherent. Operation—peptic ulcer. Partial gastrectomy
46	Hypertrophic gastritis	0.15	Filling defect ; ? carcinoma	Whole stomach seen. Carcinoma excluded. Gastritis treated medically

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
47	Hypertrophic gastritis; healed ulcer	0.2	Doubtful ulcer near stoma	Case of dyspepsia after gastro-enterostomy. Failed to see stoma. Very mild gastritis observed. Ulcer therefore improbable. Recently healed erosion seen
48	Distortion by band	0.18	Distortion; ? duodenal and lesser-curve ulcers	Lesser-curve ulcer excluded. Distortion by extra-gastric adhesions. Medical treatment
49	Gastrostaxis	—	—	Case of recurrent hæmatemesis (6 attacks in 2 years). Diffuse submucous hæmorrhages seen. (Blood-platelet count, 60,000)
50	Lesser-curve ulcer	0.06	Dubious; ? ulcer	Lesser-curve ulcer seen to be healing. Medical treatment
51	Lesser-curve ulcer	0.06	Ulcer demonstrated	Clinical suspicion of malignancy. This excluded by gastroscope. Ulcer seen to be healing. Operation nevertheless performed two weeks later. Typical peptic ulcer found half healed. (<i>See Fig. 348</i>)
52	Lesser-curve ulcer	0	Ulcer; ? carcinoma of lesser curve	Case very suggestive of carcinoma. Feeble patient. Typical peptic ulcer observed. Medical treatment. Second gastroscopy showed healing in progress. Carcinoma definitely excluded
53	Early anterior-wall carcinoma	0.09	No abnormality	Patient with vague symptoms. Ulcer observed adjacent to a swelling of the mucosa on anterior wall. Doubtful: ? ulcer; ? carcinoma. Second gastroscopy after 3 weeks showed little change. Patient discharged himself. 6 months later—a little more pain. Third gastroscopy showed extensive carcinoma. N.B.—With more experience this carcinoma could have been diagnosed extremely early. (<i>See Fig. 356</i>)
54	Atrophic mucosa	0	Dubious carcinoma of pylorus; normal stoma	Case of anæmia not responding to treatment. Short history, carcinoma suspected. Whole stomach seen. Carcinoma excluded. Normal gastro-enterostomy stoma seen
55	Hyperplastic mucosa	0.22	No abnormality	Case very suggestive of ulcer. This excluded. N.B.—Hyperplasia without gastritis

CASE No.	LESION	TEST-MEAL (1 Hr.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
56	Anæmia	0	—	Gastroscope failed: S-shaped œsophagus; instrument would not enter cardia. Ulcer seen in œsophageal wall above cardia
57	Hyperplastic gastritis; healed gastro-jejunal ulcer	—	—	Case of recently perforated acute gastro-jejunal ulcer. Sutured. Stoma clearly seen. No recurrence of ulceration observed
58	Hyperplastic gastritis; acute erosions	0.17	Ptosis; no ulcer	Marked hyperplastic gastritis and multiple erosions seen. Medical treatment. Second gastroscope showed much less gastritis, no erosions, but still marked hyperplasia of mucosa
59	Mucosal hyperplasia; healed ulcer	0.22	No abnormality	Recently healed ulcer seen on posterior wall. Hyperplastic mucosa, but very slight gastritis
60	Chronic gastro-jejunal ulcer	0.15	No evidence of ulcer	Case of repeated negative investigations of persistent symptoms after gastro-enterostomy. Chronic ulcer observed on gastroscope. Partial gastrectomy. (See Fig. 354)

I should like to express my appreciation to the Association of Surgeons of Great Britain and Ireland of the honour of being elected Moynihan Fellow for 1936 in order to undertake this work.

I wish to thank also the authorities at the London Hospital for permission and facilities to carry out the research, and the physicians and surgeons for allowing me to investigate cases under their care. In particular, I desire to express my gratitude to my chief, Sir James Walton, for his generous encouragement and for the help and advice which he has constantly given. My thanks are also due to the artist, Miss McLarty, for her patience and skill and for the care with which she has prepared the paintings.

FUNNEL-NECK DEFORMITY OF THE BLADDER

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INTRODUCTION

SINCE the days of Romberg disturbance of micturition has been recognized as one of the commonest symptoms of lesions of the spinal cord, and of *tabes dorsalis* in particular. Gowers has pointed out "that it may be only for these that the patient seeks advice." Following his introduction of the modern cystoscope, Nitze described the presence of trabeculation of the bladder wall in *tabes dorsalis*. Since then there has been considerable discussion as to the characteristic vesical findings in disease of the central nervous system. Most reports on this subject have dealt with the importance of trabeculation. This trabeculation has by many been attributed to atony and atrophy of the bladder wall, of which the internal vesical sphincter is an integral part. It might have been foreseen, therefore, that the sphincter would partake in such changes, and it comes as somewhat of a surprise that until recently relatively few observations should have been made on the appearance of the vesical sphincter in nervous disease.

On cystoscopic examination of the normal male, if the instrument is withdrawn for a short distance from the bladder so that it lies within the posterior urethra, it is impossible to see the walls of that channel, as the

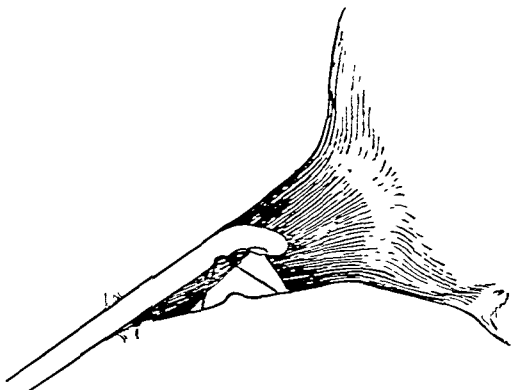


FIG. 360.—Pronounced funnel-neck deformity. Cystoscope held over verumontanum.

urethral mucosa surrounded by the internal vesical sphincter is in close contact with both the prism and the lamp of the cystoscope. In certain circumstances, however, the internal urethral orifice is flaccid, and it is then possible to withdraw the cystoscope into the posterior urethra and to obtain a clear view of its postero-inferior wall and of the verumontanum (*Fig. 360*). This condition has been variously termed 'funnel-neck deformity of the bladder', or 'funnel-shaped posterior urethra'. From consideration of the bladder musculature it appears that such a condition might arise by dilatation of the prostatic urethra following either paralysis or paresis of the internal sphincter, the whole posterior urethra thus assuming a conical shape with the base of the cone directed towards the bladder

and the apex at the triangular ligament. This explanation may be provisionally accepted as the anatomical basis of the condition.

The funnel-neck bladder must not be confused with an appearance which has much in common with it but which arises from quite a different cause. In prostatic hypertrophy the enlarging adenoma frequently dilates the internal sphincter, and the growth of the lateral lobes produces a deep cleft-like urethra at the bottom of which, if the cystoscope is held at a suitable angle, the floor of the urethra together with the verumontanum can be clearly seen. A true funnel-neck deformity can exist only in the absence of prostatic enlargement.

REVIEW OF THE LITERATURE

In 1914 Alexieff demonstrated to the Russian Urological Society a patient who from childhood had suffered from nocturnal enuresis, and in whom the posterior urethra with the colliculus seminalis was visible. Later in the same year he published his communication "On the Diagnosis of Nocturnal Enuresis." To Alexieff the gaping posterior urethra indicated a persistent weakening of the internal sphincter, and offered an explanation of the enuresis.

In the same year, and prior to the above observation, Barrington demonstrated dilatation of the posterior urethra following experimental lesions of the nervous system. He obtained cystograms of etherized female cats, and under certain conditions (following division of the pelvic nerves, of the sacral dorsal roots, or transection of the spine in the lower thoracic region) observed some dilatation of the upper part of the urethra.

In an article entitled "Prostatisme sans Prostate" Randall (1915) wrote, "By gently withdrawing the instrument with the objective posterior and the ocular well depressed, it is possible to detect relaxation of the internal vesical sphincter, which is pathognomonic of spinal cord disease, especially tabes dorsalis. The picture thus obtained is at times remarkable, and often the entire floor of the posterior urethra may be observed with the Nitze cystoscope."

So far as we are aware this is the first reference to funnel-neck deformity in the American or English literature. Randall, however, in a private communication to Natanson, does not claim originality for the observation, but states that H. H. Young first drew his attention to the phenomenon about 1910. It seems improbable that the sign was well-known at this time, as Barney in his series of articles on the tabetic bladder in 1910 makes no reference to it either in his own patients or in his review of the literature. Koll (1915) and Greenberg (1916), in articles dealing with the cystoscopic appearances of the bladder in nervous lesions, also do not comment on the phenomenon.

In 1916 Caulk and Greditzer reported their observations on the cystoscopic appearances of the bladder in fifty cases of disease of the central nervous system. Their cases included examples of tabes dorsalis, dementia paralytica, post-apoplectic conditions, tumours of the cord, and other diseases. They comment on the visibility of the posterior urethra, and, although they have also observed this condition in chronic prostatitis and seminal vesiculitis, believe that the sign is of considerable diagnostic importance. They attempted to obtain cystograms showing the continuity of the bladder and prostatic urethra, but to their surprise found a well-defined boundary at the vesical orifice and no silver solution in the urethra. These

observers were confident that a diagnosis of *tabes dorsalis* and spinal cord disease may almost always be made by cystoscopic examination, and that frequently the bladder evidence is available before any other lesions are manifest.

In another paper (1919) Caulk and Greditzer, in conjunction with Barnes, reported further cystoscopic findings in 500 cases of nervous and mental disease. They confirmed their previous belief in the diagnostic importance of a funnel-neck bladder and described various degrees of relaxation. They found that in some instances only the first part of the urethra was visible, in others the whole prostatic urethra permitted indistinct inspection, whilst in a third group there was well-marked relaxation and the whole floor of the urethra and the verumontanum could be identified. These authors regard this latter degree as a characteristic and significant finding in organic nervous disease. The other types are less convincing, and seem to occur quite frequently in the presence of a negative neurological examination, as also in certain psychoses. In patients suffering from *tabes dorsalis* the sign was present in 80 per cent of cases. In the female, dilatation of the sphincter is more difficult to identify, but the authors believe that relaxation in the presence of trabeculation suggests a nervous lesion.

In 1917 Burns published his observations on bladder changes due to lesions of the central nervous system. Twenty of his twenty-one cases suffered from *tabes dorsalis*. He noted the diminution in tone of the bladder musculature, and found the vesical orifice to be either remarkably dilated or to exhibit considerable diminution in its tonicity. The cystoscope could be drawn down into the posterior urethra and the floor of that passage together with the verumontanum could be readily observed. Burns, like Caulk and Greditzer, endeavoured to show this dilatation by means of cystograms, but found that radiographic evidence was obtained only when complete incontinence existed. Loss of ureteral tone was occasionally shown by fluid flowing up the ureter. Burns furthermore resected the posterior sacral roots in dogs and demonstrated dilatation of the internal sphincter and a funnel-shaped posterior urethra, thus supporting Barrington's observations on cats.

Several other references appear in the American literature. Braasch (1917) considers dilatation of the internal sphincter a late phenomenon in nervous disease. In conjunction with Hager (1927) he points out that cystograms in patients suffering from cord lesions frequently portray three cardinal findings: (1) A large irregular bladder outline with trabeculation; (2) Relaxation of the internal sphincter; and (3) Regurgitation of the medium into one or both ureters.

In fourteen cases of shell fracture of the spine submitted to cystoscopy, Plaggemeyer (1919) noted normal or hypertonic contraction of the external sphincter but complete relaxation of the posterior urethra, so that the verumontanum appeared to lie in the floor of the bladder. In a series of three hundred and fifty cystoscopies Cumming (1922) noted the phenomenon twelve times. These patients suffered from a variety of urinary diseases, but none was proved to have a neurological lesion. The picture of the vesical neck was similar to that seen in a neurological bladder, except that the internal sphincter tended to retain some tone so that "there is a zone of poor focus" as the cystoscope is withdrawn over the sphincteric ridge. In these cases Cumming believed that the phenomenon did not exist except with the instrument in position. He, however, agrees that the phenomenon is usually well-marked in spinal lesions.

Meyers (1926) also refers to the funnel-shaped outlet of the tabetic bladder. He attributes the relaxation of the internal sphincter to the destructive effect of tabes on the sympathetic supply to the bladder, and likens it to paralysis of the ciliospinal fibres of the sympathetic causing the extreme myosis frequently observed in this disease.

More recently Learmonth, working on the neurophysiology of the urinary bladder in man, observed that whilst sacral anæsthesia caused some dilatation of the internal sphincter, the phenomenon became more pronounced after division of the sympathetic supply. Stimulation of the presacral nerve or its lateral roots caused contraction of the internal sphincter. The dilatation which followed division of the sympathetic was not, however, permanent, and Learmonth states that "the internal sphincter may close completely. However, it can be easily opened by the advancing beak of the cystoscope and gives one the impression that it opens more easily than normal." He concludes that the ability of the sphincter to remain closed after sympathetic neurectomy proves that it must itself possess inherent tone. He refers to the fact that in the 'cord-bladder' the internal sphincter is found to be relaxed, and states that "in these cases the lesion is situated somewhere in the parasympathetic pathway. We are forced to conclude that maintenance of closure of the internal vesical sphincter depends upon its inherent tonus, which in turn depends upon the integrity of reflex fibres in the parasympathetic pathway; and that its sympathetic supply merely reinforces this closure."

In the British literature D'Arcy McCrea (1926) refers to funnel-neck deformity of the bladder, but found it only once in a series of thirty cases of tabes dorsalis. He also noted the phenomenon following sacral anæsthesia.

One of the present writers (J. B. M.) in an address to the Section of Urology of the Royal Society of Medicine (October, 1934) dealt amongst other things with funnel-neck deformity of the bladder, and the present investigation is a continuation and elaboration of certain work therein reported.

In a recent article on "Bladder Function in Spinal Injury", Watkins reports the appearance of funnel-neck deformity in lesions of the conus and cauda equina.

On the Continent considerable attention has been given in certain clinics to funnel-neck deformity. In the German literature, which of course includes many articles from Russian and Austrian sources, a fair number of references may be found. Carl Schramm in 1920 published a paper describing in detail six cases in which he had observed this phenomenon. He claimed originality for his observations, and was apparently unaware of the previous publications in both American and German literature. It is, however, noteworthy that the cystoscopic examination at which he first observed this phenomenon was carried out in January, 1912. His cases included two of spinal injuries, and one each of tabes dorsalis, disseminated sclerosis, spina bifida, and a case in which complete paresis of the bladder-closing mechanism was present but no cause could be found ("Kriegsdienstbeschädigung"). From this time the phenomenon has been referred to in the German literature as "Schrammsches Phänomen". Schramm found in many of his cases, in addition to the paralysis of the internal sphincter, a flaccid paralysis of the floor of the pelvis, atony of the sphincter ani externus, and various sensory disturbances. He concludes that the sign may appear as an early symptom of affection of the central nervous system and will frequently do so when all other symptoms are still absent. The phenomenon is, in his opinion, objective and indisputable proof of permanent

damage to the bladder-closing mechanism. In some traumatic cases where function has been re-established by recovery of the sphincter externus, gaping of the internal sphincter persists and is recognizable evidence when other neurological signs have disappeared. Schramm was so confident as to the value of the sign that in cases of legal dispute he believed incontinence of urine may be discredited in the absence of objective findings at the bladder neck. A later communication makes it doubtful if Schramm still upholds this last conclusion. At the German Urological Congress of 1926 he also stressed the fact that the phenomenon is positive only if the colliculus appears at once on withdrawal of the cystoscope but without depression of the ocular.

Pfister was the first to question Schramm's views. He could establish no relationship between dysfunction of the sphincter and visibility of the posterior urethra, and denies the diagnostic value of the sign. He states that on many occasions when the colliculus was visible, a strong contraction of the sphincteric muscle subsequently occurred. He quotes six cases of spinal trauma in which the sign was present, but in a further six cases of definite vesical paralysis following injury to the cord the sign was absent. Three cases of the deformity in which examination of the central nervous system gave no pathological findings are also mentioned.

In 1922 Oswald Schwarz (Vienna) published a severe criticism of Schramm's views. Stating that he himself had observed a funnel-neck urethra in a case of tabes as early as 1920, he admits that the sign may occur, but denies to it any diagnostic significance. In proof of his assertion he points out that the phenomenon is frequently absent in cases of unmistakable nervous disease, and that even when observed it is extremely inconstant, being present on one examination and absent on another; indeed it may appear and disappear during a single cystoscopy. Schwarz, like Pfister, has observed the phenomenon when no lesion of the central nervous system could be demonstrated. He maintains that the longer the cystoscope remains in the bladder the more likely is the phenomenon to develop, and especially if the ocular end of the instrument is unsupported will its vertical drag cause a dilatation of the sphincter and the artificial production of the phenomenon. In support of these arguments he states that in his experience normal ejaculation (which depends on the internal sphincter for the forward direction of the sperm—see pp. 507, 513) may occur in those showing the phenomenon. The base of the bladder, as seen on a cystogram, also exhibits a normal outline. Oswald Schwarz maintains that the symptoms of disturbed bladder function point to hypertrophy of the internal sphincter rather than to its relaxation. The dilatation of the sphincter may be reflex rather than mechanical in origin, the irritation of the posterior urethra by the cystoscope possibly inducing Barrington's fourth reflex. He draws an analogy between the state of affairs occurring at the internal sphincter and the condition obtaining at the lower end of the œsophagus in cardiospasm, and points out that in the latter the passage of a sound produces relaxation of the sphincter.

Goldenberg (1924) confirmed Oswald Schwarz's views regarding the inconstancy of the sign and the possibility of its production by depression of the cystoscope. He also mentions maximal distension of the bladder and sacral anæsthesia as additional means of obtaining dilatation of the internal sphincter. In general he denies the value of the sign, but considers that it may be of some significance when combined with trabeculation and residual urine.

Against these adverse criticisms O. A. Schwarz (Berlin) and Perlmann appear as protagonists. They regard the sign as having great diagnostic value, and consider that it may appear as the only evidence of nervous disease. In one publication O. A. Schwarz quotes fifteen cases of tabes and cerebrospinal syphilis, in only one of which the sign was absent. He was particularly impressed by patients in whom, although there had been quite indefinite disturbance of micturition or none at all, the sign was found positive, and in whom subsequent careful neurological examination showed the existence of tabes. Schwarz also observed patients with a visible colliculus who were suffering from neurasthenia and in whom there was no demonstrable organic lesion. He expresses no view regarding the occurrence of the phenomenon in the healthy individual.

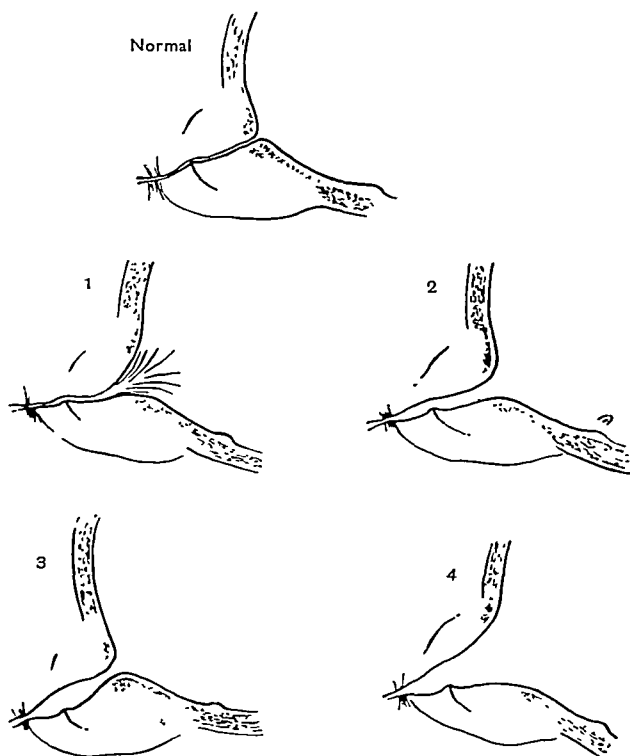


FIG. 361.—Types of deformity—Moro's classification. (See text.)

In view of these contradictory opinions Moro in 1927 investigated the material in his clinic and scrutinized the relationship existing between the trigone and posterior urethra in all cystoscopies. He differentiated four grades or degrees of dilatation (Fig. 361):—

Type I.—Radial streaking in the region of the internal urethral orifice, with more gradual transition of the bladder into the posterior urethra replacing the normal sharp sphincteric margin.

Type II.—The floor and side walls of the posterior urethra at the lower end are just visible and the colliculus is indistinctly seen as it lies close up against the prism.

Type III.—The field of view is dark in its proximal part, but as the cystoscope is further withdrawn it enters a relatively spacious, spindle-shaped chamber in which the illumination is bright. On the floor of this chamber the colliculus is seen, together with its surrounding vallecule.

Type IV.—The colliculus lies in full view in a brightly-lit channel, which allows some movement of the instrument to either side. The passage between the posterior urethra and the bladder is so wide that the posterior urethra appears as a funnel and the normal angulation at the point of transition fails (*see Fig. 360, p. 501*).

Moro placed diagnostic value only on *Types III and IV*. He did not invariably find the sign either in tabes or trauma. His conclusion was that when the nerve-supply of the bladder is damaged, gaping of the posterior urethra with visibility of the colliculus may occur. Once developed the phenomenon is constant, and may in traumatic cases persist as the only objective sign of preceding nervous injury. The sign is not, however, pathognomonic of nervous disease, as it may be artificially produced by tiring the sphincter, and thus may appear in healthy men. In such circumstances the sign is inconstant and may come and go during one and the same examination. As the sign, however, may indicate incipient nervous disease, it calls for a complete neurological overhaul.

In the same year (1927) Natanson also published his findings on the phenomenon, and he summarizes his views by stating: "It is certain that it occurs in patients with a healthy spinal cord, especially neurasthenics, but it is found so frequently in spinal and cerebral diseases (almost regularly in tabes and enuresis) that this association cannot be disregarded." He also admits that the establishment of a 'colliculus phenomenon' may, especially in tabes, be the first objective sign of existing disease of the spinal cord, and, in view of the importance of an early diagnosis, he sees no sufficient reason to deny the practical importance of the phenomenon.

In 1929 Fessler and Fuchs (Vienna) investigated sexual function in relation to funnel-neck deformity of the bladder. They pointed out that whilst in the normal male some of the secretion expressed by prostatic massage flows down the urethra and appears at the external meatus, the remainder runs back into the bladder and is subsequently voided in the urine. The proportion entering the bladder depends on the tone of the internal sphincter, and is maximal when this structure is paralysed. Five tabetics, in whom no secretion appeared at the external orifice following prostatic massage, exhibited the phenomenon on cystoscopic examination. Fessler and Fuchs also investigated fourteen male tabetics who, despite the well-known deterioration occurring in this disease, were still capable of sexual intercourse. Three gave a clear history that, although intercourse and orgasm were normal, there was a complete absence of ejaculation, the flow of semen undoubtedly passing back into the bladder.

The Russian literature contains several important papers on funnel-neck deformity of the bladder. The largest series so far examined is that of Epstein, Juschelevsky, and Michelson, who announced their results to the Russian Urological Association in 1928. They examined five hundred patients, two hundred of whom were known to be suffering from nervous disease; the remaining three hundred constituted the ordinary cystoscopic material of their clinic. Of the healthy men examined the vast majority showed no funnel-neck deformity. Attempts to produce the sign by movements of the cystoscope were also negative. The investigation of

the first group—namely, patients known to be suffering from disease of the central nervous system—appears to have definitely established that a funnel-neck depends not so much on the variety of the disease as upon its situation. The majority of cases showing it in well-marked development were instances of lesions of the spinal cord. In male patients suffering from *tabes* the sign was present in 90 per cent of cases, in syphilitic myelitis in 80 per cent. Examples of spina bifida, though not occurring in any numbers among their cases, were found to exhibit a high proportion of funnel-necks, whilst quite a few patients in whom the sign was positive, and who yet showed no central nervous disease, were found by radiology to have some degree of spina bifida which was otherwise unrecognizable. These authors conclude that a funnel-neck urethra is an early sign of nervous disease, and that its presence indicates an organic lesion or developmental defect. They also hold it to be unlikely that a funnel-neck is ever discovered in the healthy male, and in their opinion it is doubtful whether it is to be found in uncomplicated functional disturbance. These authors believe that dilatation of the internal sphincter may also be observed in women, although the phenomenon is never so pronounced as in the male. Their series of patients with nervous diseases included fifty women.

Halperstein (1928) found the sign on twenty-two occasions (*tabes* 12, spinal trauma 3, spina bifida 2, etc.) and attributed great value to it.

In another Russian publication, Dunajewsky and Michejew (1931) allow little diagnostic value to this sign, and have found it positive in a much smaller number of cases. Examining forty-two cases of *tabes* they found it present in but 16.6 per cent. Their observations on two hundred men, in whom there was no evidence of organic nervous disease and in five of whom the sign was present, led them to the conclusion that the sign could be artificially produced by the pressure of the cystoscope, and that it might be observed in healthy or neurasthenic patients. They considered trabeculation to be a more frequent and definite sign of nervous disease.

Recently Franz Gáspár (Budapest) has drawn attention to the fact that by using local anæsthesia it is possible to obtain complete relaxation of the internal vesical sphincter and to carry out with a cystoscope a complete inspection of the posterior urethra. He maintains that a better view is thus obtained than by using a posterior urethroscope. A catheter is inserted into the bladder and then withdrawn until its tip lies between the internal and external vesical sphincters. A local anæsthetic is then slowly injected over a period of half an hour, and a further half-hour is allowed to elapse before cystoscopy. Using novocain (1 per cent) or percaïne (1–1000) full dilatation of the posterior urethra was obtained in eighteen out of twenty cases.

Short references to funnel-neck deformity of the bladder have also been made by Gagstatter, Schwarzwald, Young, Heplar, von der Becke, and Deutsch.

CLINICAL OBSERVATIONS AND CYSTOSCOPY

From a survey of the literature it is evident that the significance of funnel-neck deformity as a sign of nervous disease is by no means decided. We have endeavoured, therefore, to obtain an independent view of the incidence and diagnostic importance of this sign. Our inquiry concerns:—

1. Patients definitely suffering from nervous disease.
2. The frequency of the sign amongst urological patients who exhibit no evidence of nervous disease.

In examining the vesical neck for the sign, the fenestra of the cystoscope is first directed towards the trigone of the bladder and is then gradually withdrawn into the urethra. During this manœuvre it is necessary to depress the ocular end of the cystoscope slightly, otherwise the condition may escape observation. Only the floor and the lower portions of the lateral walls of the urethra can be brought into view. If the instrument is rotated, the field of vision becomes dark as soon as the upper portion of the side wall is reached, and even by manœuvring the instrument we have never been able to get a satisfactory view of the upper portion of the urethra.

The sign has been regarded as positive only when it has been recognized immediately on withdrawal of the instrument and has remained constant during the examination. Straining to micturate produces a temporary physiological dilatation of the posterior urethra.

To standardize our observations and facilitate comparison, we have adopted the classification of Moro in stating our results (p. 506 and *Fig. 361*). This classification includes all recognizable degrees of the phenomenon. In practice there is difficulty in distinguishing with certainty *Types I* and *II* of this classification from the normal. Hence we, like Moro himself, regard these grades as unreliable, and although we have used them in classifying some of our material, we do not attach any clinical significance to them, and regard only *Types III* and *IV* as constituting a funnel-neck deformity.

I. OBSERVATIONS ON PATIENTS WITH NERVOUS DISEASE

Our material consisted of 17 cases of tabes dorsalis, 5 cases of general paralysis of the insane, 2 cases of cerebrospinal syphilis including one of Erb's syphilitic spinal paralysis, 3 cases of disseminated sclerosis, and 1 of 'spastic paraplegia'. These patients were not chosen on account of the existence of urinary symptoms, but represented such material as was available for examination. The only selection we allowed ourselves was the rejection of cases in which complications would tend to alter the appearance of the bladder-neck. Infection is one such complicating factor, and accordingly all who showed evidence of cystitis, either by a dirty urine or on cystoscopy, were rejected. In practice this did not involve the exclusion of any large number, as the infrequency of this condition, even in the presence of fairly large amounts of residual urine, has been surprising. Enlargements of the prostate were also excluded.

A viewing cystoscope was used for the examination, and, recognizing the susceptibility of the bladder to infection in central nervous disease and the importance of keeping it uninfected, the most rigid asepsis was maintained. No anæsthetic of any type was used. This allowed judgement of the degree of pathological anæsthesia, and entailed little or no hardship to the patients, as in most of them urethral sensation was either diminished or almost absent. The residual urine was measured in most instances, the average amount present being 250 c.c.

Examination of the Vesical Orifice.—This revealed a funnel-neck in a high proportion of our patients. Thus, of the 17 tabetics, only 3 showed a normal orifice. The examples of general paralysis and of cerebrospinal syphilis all showed the deformity. The sign was absent in those suffering from disseminated sclerosis and spastic paraplegia, except for one example in the former disease. In this patient it was necessary to use a local anæsthetic (percaine), and this result must therefore be accepted with scepticism.

The bladder was filled in the first instance with 200 c.c. of fluid. With this amount, sagging of the vesical wall often showed that the bladder was obviously not distended and was incapable of contracting down on its contents. With such a degree of distension several cases did not exhibit a funnel-neck deformity, though when the bladder was filled to its individual capacity the sign was found fully developed. The filling was never carried to the point of causing discomfort, and in most patients much more fluid might have been introduced. This fact accounts for the occurrence of two figures attached to several of the cases in our own table of results (*Table I*, p. 512). The first figure indicates the appearance when using 200 c.c. of fluid, the second that with the bladder more fully distended. The dilatation of the posterior urethra was extremely wide in many, the trigone appearing to merge with the floor of the prostatic urethra without the intervention of a dividing rim. Excellent views were obtained of the urethral crest terminating in the colliculus, the latter projecting from the floor of the urethra as a pale pink hummock bounded on either side by the vallecule. The openings of the prostatic utricle and of the ejaculatory ducts were often recognized on the surface of this structure. Occasionally the urethra was dilated for a short distance below the colliculus. Amongst patients exhibiting *Type III* of the deformity, the dilatation of the mid-section of the prostatic urethra appeared as great as that observed in the more advanced grade, the difference between the two varieties being the constriction at the vesical orifice of the former.

The retrogression and final disappearance of the phenomenon following anti-syphilitic treatment, which Burns observed in his series, were not noted by us.

The Bladder Wall.—The general impression gained from observation of the bladder wall in the patients suffering from syphilitic disease of the nervous system was one of atony and atrophy. The vesical wall as a rule appeared extremely thin, and intestinal movements were observed with great frequency. Trabeculation was present in 15 cases. In 12 of these it exhibited the characteristic fine and delicate appearance described by Thomson-Walker. In the other 3 bladders the degree of trabeculation was either insignificant or atypical. The ureteric and inter-ureteric bars frequently lacked their normal fleshiness, and as the large retro-trigonal recess fell away steeply behind them, they appeared to be unduly projected into the bladder.

Cystography.—Many attempts were made to obtain cystograms showing the funnel-neck deformity. With four exceptions (*Cases 2, 7, 10, 22*), however, these efforts ended in failure, the bladder showing the normal smooth basal contour, with no opaque solution in the posterior urethra. Irregularity in the shape of the bladder and the coarser varieties of trabeculation are shown by this means. Twice the opaque fluid regurgitated up one or other ureter.

The relative infrequency of cystograms showing a funnel-neck deformity seems remarkable, but is in accordance with the findings of other observers (Caulk and Greditzer, Otto Schwarz, Burns). The last-mentioned author obtained outlines of the prostatic urethra only when complete incontinence existed.

The technique of cystography consisted in filling the bladder with the opaque solution through a catheter, which was withdrawn prior to exposure of the film. The volume of fluid employed depended on the previously determined bladder capacity. Potassium iodide (2.5 per cent) and silver iodide (5 per cent emulsion) were both tried as opaque solutions, the latter being reputed to be less irritant than

the former. Each of these solutions, however, was liable to cause discomfort and a desire to micturate. Efforts to maintain the bladder pressure at a low level were thus frequently frustrated, and whilst in some instances large volumes of fluid were introduced at a pressure below 10 cm. of water, in others small volumes (200 to 250 c.c.) caused discomfort, and the bladder pressure, despite a period allowed for the establishment of equilibrium following filling, remained at 30 to 40 cm. of water.

Guyon, as long ago as 1901, showed that following section of the *nervi erigentes* and injection of an irritant (AgNO_3) the bladder would still contract and expel part of its contents, and from this concluded that under certain conditions the bladder musculature was independent of all medullary influence. Head and Riddoch have described complete automatic micturition, consisting not only of contraction of the bladder but of relaxation of the sphincter, following destruction of the sacral roots by trauma. Denny-Brown and Graeme Robertson also believe that automatic micturition may occur following complete sacral lesions, and that the internal sphincter relaxes reciprocally with contraction of the detrusor.

These observations suggest that an artificially produced funnel-neck deformity might be shown on cystograms obtained with an irritant solution, either by the occurrence of contraction of the detrusor and reciprocal relaxation of the internal sphincter, or by contraction of the detrusor raising the intravesical pressure sufficiently to force the internal sphincter. Despite this, in the above series, the deformity was but rarely found on cystograms. Even when a positive cystogram is obtained it must be determined whether it faithfully depicts an existing condition or is artificially produced. In illustration of this view *Case 22* may be cited. Cystography performed on this man with a potassium iodide solution showed a large funnel-neck deformity (*see Figs. 364-367, pp. 522, 523*), whereas subsequent excretion cystography failed to demonstrate any deformity of the bladder neck. The relatively high intravesical pressures recorded in some instances prior to the withdrawal of the catheter and the absence of opaque fluid in the posterior urethra on subsequent cystograms also throw doubt on the pressure usually stated to be sufficient to force the internal sphincter.

It thus appears that whilst ascending cystography may satisfactorily serve most ordinary purposes it is unsuitable for observations on the bladder neck. So many complicating factors are introduced that unless the bladder pressure has been low prior to the withdrawal of the catheter little value can be placed on the evidence. Excretion cystography offers material advantages, as the fluid is less irritating and mechanical interference with the posterior urethra is avoided.

Relationship between Urinary Symptoms and Funnel-neck Deformity.

—The order of onset of urinary disturbance in tabetics is variable, but in our experience the following would appear to be the most usual course: Diminution or loss of the normal desire to micturate leads to infrequency of the act, urination taking place only once or twice daily. The quantities passed are then necessarily large. Coincidentally the patients may experience difficulty in starting the act and the stream may lack force and be intermittent. The whole of the vesical contents are not expelled and residual urine commences to collect. Signs of incontinence may appear at this stage, or may have shown themselves at an earlier time; in some instances they constitute the initial symptom. At first incontinence may be limited to occasional nocturnal enuresis, or dribbling following micturition. Sudden

exertion may cause the escape of small jets of urine. With progression of the disease the volume of residual urine tends to increase, and abdominal straining becomes more and more necessary to empty the bladder. The incontinence also may become more pronounced, a distended bladder being frequently accompanied by either intermittent or almost continuous dribbling of urine. Active incontinence was not observed amongst the tabetics, but occurred once in a case of spinal syphilis and again in a single instance amongst the general paralytics. No enormously distended bladders occurred in any of our patients.

Difficulty in micturition, though frequently present in minor degree, has not been a troublesome symptom, and complete retention has occurred in the early stages but once. One other patient complained of attacks of acute retention, but emergency catheterization never discovered more than three or four ounces of urine in his bladder. There have been no instances of urethral anæsthesia so complete that micturition has been recognized only by wetting of the clothes.

Table I.—FUNNEL-NECK DEFORMITY IN NERVOUS DISEASE

DISEASE	PRINCIPAL URINARY SYMPTOM	CASE AND DEGREE OF DEFORMITY
1. Tabes dorsalis (17)	1. No definite symptoms (3) ..	Case 2 (IV); Case 3 (IV); Case 9 (0 → IV)
	2. Absence of desire to urinate (3). Infrequent micturition with passage of large amounts of urine	Case 1 (I → IV); Case 4 (II → IV); Case 16 (IV)
	3. Incontinence, dribbling, nocturnal enuresis (8)	Case 5 (III or IV → IV); Case 6 (III); Case 7 (IV); Case 8 (I or II); Case 10 (II → IV); Case 12 (IV); Case 14 (IV); Case 17 (II → IV)
	4. Difficulty (2)	Case 13 (II → IV); Case 15 (0)
	5. Sensation of distension without residual urine (1)	Case 11 (0)
2. G.P.I. (5) ..	1. No symptoms (2)	Case 19 (III); Case 20 (II or IV)
	2. Dribbling urine (2)	Case 18 (III → IV); Case 22 (IV)
	3. Active incontinence (1) ..	Case 21 (IV)
3. Cerebrospinal syphilis (2)	Active incontinence	Case 23 (IV)
	Incontinence	Case 24 (III)
4. Disseminated sclerosis (3)	—	(0); (0); (III)
5. Spastic paraplegia (1)	—	(0)

Table I shows that there is no real difference in the incidence of funnel-neck deformity amongst those complaining of incontinence and those who have no such trouble. The internal sphincter normally retains the urine within the

bladder and is possibly responsible for continence during sleep, the external sphincter relaxing as do all voluntary muscles. It might therefore have been anticipated that paresis of the internal sphincter would play a part in the nocturnal incontinence so often seen in tabes. On the other hand, the internal sphincter is usually destroyed or out of action following the operation of prostatectomy, and yet these patients are not incontinent at night. Likewise the internal sphincter may be destroyed by trauma and yet continence be complete.* *The external sphincter in the normal male is therefore quite capable of assuming this function of the internal sphincter. The onset of incontinence in tabes dorsalis thus suggests not only that the internal sphincter is atonic, but that the external sphincter is undergoing similar changes. The discovery of a funnel-neck deformity does not necessarily predict the onset of incontinence.*

Sexual Function.—Realizing that the internal sphincter is the barrier which prevents sperm passing back into the bladder during ejaculation and that weakness of this muscle might lead to a failure of this function, we inquired in a few cases regarding emission. It was not, however, possible to investigate the sexual function of all patients in this series. Of the tabetics, 2 reported that orgasm without ejaculation occurred, whilst on the other hand 2 others who exhibited the deformity were emphatic that a normal emission of semen occurred.

DISCUSSION

From the above observations it is evident that in syphilitic disease of the central nervous system the cystoscope will almost invariably reveal a funnel-neck deformity of the bladder. The most reasonable explanation of the phenomenon is weakness of the internal vesical sphincter. The appearance suggests that the bladder and dilated posterior urethra form one cavity, but cystography in the majority of instances refutes this suggestion, and establishes beyond reasonable doubt that the deformity usually exists only when the cystoscope is in position, and the question therefore arises whether it may not be artificially produced. Depression of the cystoscope has been advanced as a possible cause, but endeavours to produce the phenomenon by this means in the healthy male with a normal posterior urethra have met with little or no success, and even the steepest depression has never produced a *Type III* or *IV* deformity. The cystoscope alone, therefore, is insufficient to produce the phenomenon, and the sign must primarily indicate weakness of the internal sphincter.

Conversely, the absence of a resultant shadow on cystography shows that the internal meatus is closed when no instrument occupies it, and the dilatation seen cystoscopically must be due to mechanical separation of the weakened sphincter. This closure so frequently observed on cystograms may be due on the one hand to tone remaining in the internal sphincter sufficient to close the urethra, or on the other hand to elastic tissue, of which an abundance is known to exist in this situation, performing the task. Whichever explanation is true, and it seems probable that a combination of forces operates, the closure of the bladder neck must be quite weak, as the lightest movement of the cystoscope suffices to separate the urethral walls in a manner usually impossible in the normal unanæsthetized male urethra. That elasticity of the tissues around the bladder neck may act in this way

* See two cases reported by one of us (J. B. M.), *Proc. Roy. Soc. Med.*, 1934, xxviii, 1.

is suggested by the fact that in complete incontinence due to paralysis of the internal and external sphincters the outflow of the urine is not continuous. Small quantities collect during periods of inactivity and are expelled only when movement temporarily raises the intravesical pressure. It is also well known that urine does not escape from the bladder following death (no matter what the position of the body). At the Urological Congress of 1933 Weijtlandt quoted Guthrie (1834) and Barkow as regarding the sphincter not as a muscle but as elastic tissue, whilst Kenneth Walker also drew attention to the importance of elastic tissue in this situation. Watkins quotes Schwarz and Dennig as having made similar observations.

The nerve-supply of the bladder is derived from the sympathetic and sacral parasympathetic system. Much diversity of opinion exists concerning the exact distribution and function of each system. In man, according to Learmonth, the sympathetic supply arises from the first, second, third, and fourth lumbar ganglia on either side, and the intermesenteric plexus, and proceeds to the bladder by the presacral and hypogastric nerves. The segmental level of the connector cells is not known with accuracy, but is frequently attributed to the first and second lumbar segments. The afferent sympathetic fibres enter the spinal cord between the ninth thoracic and fourth lumbar segments. The sacral autonomic fibres (afferent and efferent) arise from the second and third sacral segments. The prostatic urethra and external sphincter are supplied by the pudendal nerve, which springs from the third and fourth sacral segments. For the purpose of this discussion it is only necessary to consider the function of the sensory paths. It is probable that the afferent sympathetic fibres convey painful impulses and also some information regarding distension. The main sensory path is, however, the parasympathetic system. The degree of distension of the bladder, tactile and thermal stimuli, and some painful impulses are conveyed by this route. Its fibres also include the vast majority of those concerned with the micturition reflexes, and a proportion of sensory fibres from the posterior urethra.

The primary lesion in tabes is a syphilitic inflammation of the posterior nerve-roots central to the ganglion, with subsequent degeneration of the efferent neurons and sclerosis (Richter). These changes appear first in the lower lumbar and upper sacral regions. In time complete atrophy of the ganglion cells and of the peripheral portions of the sensory nerves may occur. The afferent autonomic fibres are affected in this degeneration. The efferent fibres and ganglion cells probably escape.

Since the tabetic lesion commences in the lumbosacral region the main sensory tracts from the bladder are impaired at an early date. Though gradually losing its sensory supply, the bladder remains in contact with the motor centres of the brain and spinal cord. The hypogastric ganglia and vesical plexuses are also probably unaffected. The tabetic bladder, therefore, differs in important respects from the 'cord-bladder' described by Head and Riddoch, Holmes, Denny-Brown and Robertson, etc. In the cord-bladders described by these authors there are two main groups. In the one a transverse lesion of the cord leaves the sacral reflexes complete; in the other a conus or cauda equina lesion destroys the sacral connections, but its sympathetic supply and its connections with the hypogastric and vesical ganglia remain intact.

In our own experience the relaxation of the internal vesical sphincter occurring in tabes appears to be part of a general loss of tone of the bladder musculature. The collection of residual urine, the incompetence of the vesical sphincters, and

the atrophic appearance of the vesical wall all suggest atrophy. Many observers (Frankl-Hochwort, Böhme, etc.) have ascribed the trabeculation to hypertrophy. Our own view agrees with that of Thomson-Walker, who attributes the characteristic appearance to atrophy of the bladder wall. The tone of smooth muscle is known to be much more independent of nervous reflexes than that of striated muscle, but the gradual and progressive loss of detrusor and sphincteric tone occurring in tabes suggests that the reflex arc is of considerable importance. It appears probable that the first result of the loss of proprioceptive impulses is a diminution of detrusor tone, which, apart from any loss of conscious sensation, is itself sufficient to account for the early onset of too infrequent micturition. Concurrently atonia of the internal sphincter develops and a funnel-neck deformity becomes visible on cystoscopic examination. The loss of tone in the detrusor and sphincters would appear to be progressive, the bladder, if uncomplicated by cystitis, tending to become a flaccid organ. Loss of the local sphincteric reflexes may cause some sphincteric difficulties, but except for the attacks of acute retention occurring at the onset of the disease (at which stage the heightened skin reflexes suggest the possibility of sphincteric spasm) there does not seem to be any evidence for assuming abnormal spasticity of the sphincters as a cause of retention. Whether retention or incontinence shall predominate depends on the relative atonicity of the detrusors and sphincters.

The results of section of the posterior sacral nerve-roots in animals provide an interesting comparison. In the dog, Dennig and Burns have shown the resulting disturbances of micturition to be almost identical with those of the advanced tabetic. Barrington, however, in similar experiments on cats, observed relaxation of the bladder with retention of urine, which if unrelieved proceeded to overflow incontinence. Efforts to express urine from the bladder in these cats were met by strong and sustained resistance. Cystograms showed the internal sphincter dilated and the fluid held at the external sphincter.

CASE REPORTS OF NERVOUS DISEASE

Case 1.—W. P. Aged 47. Tabes dorsalis.

Primary syphilis twenty-six years ago. No immediate treatment. Diagnosed as tabes dorsalis eight months ago. History of 'tiredness' but no other symptoms. Pupils now pin-point and show no reaction to light. Knee- and ankle-jerks absent. No loss of sensation. Wassermann and Kahn tests positive. Received eight months' treatment for neurosyphilis prior to examination.

URINARY SYMPTOMS.—Passes large quantities of urine. Normal desire to micturate; good stream; no interruption. F.=D.2-3 N.O.* No retention. No incontinence. No enuresis. No intercourse with wife for nine months. No emission of semen.

CYSTOSCOPY.—Passed 1320 c.c. urine prior to examination. Residual urine 200 c.c.; clear. Bladder filled with 200 c.c. fluid. Bladder wall in folds, obviously not distended. No cystitis. Trabeculation, especially in the roof of the bladder, very fine in type. Intestinal movements observed through the bladder wall. Bladder neck showed questionable degree of dilatation (*Type I*). Bladder further distended but without causation of any discomfort. Funnel-neck urethra developed (*Type IV*).

Case 2.—F. H. Aged 60. Tabes dorsalis.

Date of infection with primary syphilis unknown. Has suffered from upper abdominal pain for three to four years. Repeated X-ray examination has revealed no pathological lesion of the intestinal tract. Diagnosed as tabes dorsalis six months ago. Wassermann and

* For simplicity of recording frequency of micturition the above abbreviation has been adopted. Thus in the present example F.=D.2-3/N.O indicates that micturition has occurred two to three times during the day and not at all at night.

Kahn tests positive (+ +). Had received six months' treatment for neurosyphilis prior to examination.

URINARY SYMPTOMS.—No urinary symptoms. Normal desire to micturate. No dribbling. No enuresis.

CYSTOSCOPY.—Urethra almost anæsthetic. Residual urine 30 c.c.; clear. Bladder filled with 200 c.c. fluid. No cystitis present. Beautiful fine and coarse trabeculation. Fully developed funnel-neck deformity (*Type IV*). Colliculus well seen in centre of a large illuminated cave. Urethra also considerably dilated below colliculus.

CYSTOGRAPHY.*—Cystogram shows a small bladder with well-marked trabeculation. The funnel-neck deformity is also shown. Some opaque fluid regurgitated up right ureter.

Case 3.—R. G. Aged 42. Tabes dorsalis.

Gonorrhœa nineteen years ago. Complaining of lightning pains for past five years. Pupils small, unequal, and fixed. Knee- and ankle-jerks absent. No loss of sensation over limbs. Blood Wassermann test positive + +. Cerebrospinal fluid shows 5 lymphocytes per c.mm.; Wassermann test +; Lange's colloidal gold curve 5 5 5 4 3 2 1 0 0 0.

URINARY SYMPTOMS.—Normal desire to micturate. F.=D.2-3/N.O-1. No difficulty. Never retention. No dribbling. No nocturnal enuresis. Married; normal emission.

CYSTOSCOPY.—Residual urine 15 c.c. Bladder distended to 200 c.c. Mucosa normal; no trabeculation. Well-marked funnel-neck deformity of bladder (*Type IV*). Colliculus and prostatic utricle well seen; urethra dilated for an inch below this. Bladder refilled with 400 c.c. fluid. No discomfort. No further dilatation.

CYSTOGRAPHY.—The cystogram (*Fig. 362*) shows trabeculation and a funnel-neck.

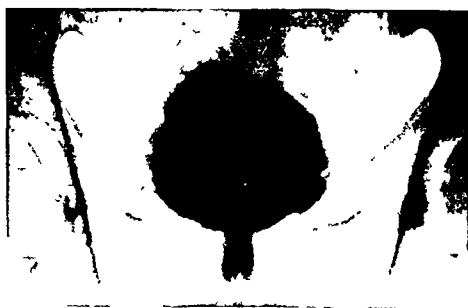


FIG. 362.—Case 3. Tabes. Cystogram showing well-marked trabeculation and a funnel-neck deformity.

Case 4.—T. J. L. Aged 58. Tabes dorsalis.

Contracted primary syphilis thirty-nine years ago in Hong Kong. Received mercurial treatment. During 1916 was invalided out of the Navy suffering from tabes dorsalis. Since then has been troubled with lightning pains and ataxia, but otherwise has remained well. At present his pupils are small, equal, and pin-point. The knee- and ankle-jerks are absent. Sensation is diminished over the legs and saddle area.

URINARY SYMPTOMS.—Infrequent micturition; F.=D.3-4/N.O. Desire somewhat diminished. No retention. No dribbling. Nine years ago used to wet his clothes, but never now. No nocturnal enuresis.

CYSTOSCOPY.—Residual urine 15 c.c. Urethra almost anæsthetic. Bladder filled with 200 c.c. fluid. No cystitis. Fair degree of trabeculation; coarse rather than fine. Internal urethral orifice appeared patulous (*Type II*). Bladder distended to 650 c.c. fluid without discomfort. Funnel-neck urethra (*Type IV*) developed. Good view of colliculus.

Case 5.—A. B. Aged 31. Tabes dorsalis.

Primary syphilis fifteen years ago. No immediate treatment. Two years ago sought medical aid on account of shooting pains in his thighs and weakness of right leg. Diagnosis

* Cystograms have been made in most cases, but were generally negative. A note has been made only where a funnel-neck was observed.

of tabes dorsalis established. Wassermann and Kahn test $++$. Cerebrospinal fluid showed 10 cells per c.mm.; Wassermann test $+$; Kahn test $++$; Lange's colloidal gold test 00123321000. At present shows marked ataxia. Pupils dilated; no reaction to light. Knee- and ankle-jerks absent. Sensation markedly diminished over legs and saddle area.

URINARY SYMPTOMS.—Retains his urine for an unusual length of time, then passes a large amount. No difficulty. No straining. Good stream. F.=D.4/N.o. May dribble a little after urinating. No enuresis.

CYSTOSCOPY.—Penile hypospadias. Urethral and bladder sensibility practically absent. Residual urine 240 c.c. Bladder filled with 200 c.c. fluid. No cystitis. The internal urethral orifice appeared patulous. This formed a loose waist between the bladder and a widely dilated prostatic urethra, in which the colliculus was clearly seen (*Type III* or *IV*). The bladder was then distended to 600 c.c. The funnel-neck deformity became more prominent (*Type IV*).

Case 6. P. B. Aged 56. Tabes dorsalis.

Date of primary syphilis unknown. Sought medical treatment fifteen months ago on account of pain in left side and dribbling of urine. Diagnosed as a case of early tabes dorsalis. Wassermann test (blood) positive ($++$). Pupils now small; no reaction to light. Knee-jerks present, ankle-jerks absent. Sensation is poor over the legs and perineum, especially over saddle area. Feels a pin but unable to distinguish the point from the head. Patient has now received a year's antisyphilitic treatment.

URINARY SYMPTOMS.—Three years ago, for a period of three to four months, suffered from nocturnal enuresis and slight dribbling incontinence during day. One year ago complained of dribbling incontinence but states that this symptom no longer troubles him. At present feels a desire to micturate, but does not think the sensation is as acute as previously. At times experiences a little difficulty in commencing micturition. Good stream; no interruption. Never retention.

CYSTOSCOPY.—Urethral sensibility very slightly impaired; feels pull on ligaments. Distension usual 200 c.c. No cystitis. Very many extremely large blood-vessels. Very marked trabeculation present, which, excepting the trigonal area, extended over the whole of the bladder including the apex. At first the bladder neck appeared normal, but on withdrawing the cystoscope slightly an illuminated cavern was entered, in which the colliculus was well displayed (*Type III*).

Case 7.—J. B. Aged 52. Tabes dorsalis.

Primary syphilis eleven years ago. Treatment for one week. Attended urological clinic complaining of difficulty in micturition. 'Shooting pains' down back of thighs for several years. Ataxic gait. Romberg's sign positive. Pupils small; no reaction to light. Knee- and



FIG. 363.—Case 7. Tabes. Cystogram showing bladder half filled with potassium iodide solution. Pressure prior to withdrawal of catheter was below 5 cm. of water. Large smooth bladder showing funnel-neck deformity and regurgitation of fluid up left ureter.

ankle-jerks absent. No real loss of sensation demonstrated. Wassermann and Kahn tests (blood) positive (+ +). Cerebrospinal fluid: Wassermann—; Kahn \pm ; Meinicke \pm .

URINARY SYMPTOMS.—Difficulty of micturition. Strains to urinate, so much so that his hæmorrhoids bleed. Passes urine infrequently; F.=D.2-3/N.o-2. At times does not feel his urine flowing. Has to wear a cloth to save his clothes.

CYSTOSCOPY.—Urethral sensation diminished. Residual urine 480 c.c. Bladder filled with 200 c.c. fluid. Obviously not distended. No cystitis. Beautiful fine trabeculation. Mucosa has an atrophic appearance. Funnel-neck urethra (*Type IV*). Excellent view of verumontanum. Bladder filled with 800 c.c. fluid. No discomfort whatever. Fine trabeculation and atrophic appearance of mucosa very marked. Funnel-neck urethra unchanged.

CYSTOGRAPHY.—Cystogram (400 c.c.) showed no trabeculation; funnel-neck demonstrated (*Fig. 363*). Some opaque fluid escaped up L. ureter. With 600 c.c. the vesical pressure was still below 5 cm. water.

Case 8.—W. S. Aged 58. *Tabes dorsalis*.

Date of primary syphilis unknown. Four years ago a left orchidectomy was performed for gumma of left testis. A diagnosis of *tabes dorsalis* was also made at this time. Patient subsequently underwent a course of antisyphilitic treatment, but attended irregularly. Condition now very much worse. Walks with great difficulty; tabetic gait. Romberg's sign well-marked. Pupils unequal and do not react to light. Knee-jerks absent. Sensation diminished from waist down; loss almost complete in saddle area.

URINARY SYMPTOMS.—Nocturnal enuresis for three to six months. Can pass urine voluntarily but occasionally dribbles slightly. No retention. No frequency. Double incontinence of urine and fæces first noticed three months ago during sleep.

CYSTOSCOPY.—Residual urine 300 c.c. Urethra almost anæsthetic. Bladder distended with 200 c.c. fluid. Bladder mucosa injected. Some trabeculation of bladder; not fine type usually described as typical. Internal urethral orifice somewhat patulous (*Type I or II*). No well-marked funnel-neck deformity.

Case 9.—J. A. N. Aged 58. *Tabes dorsalis*.

Date of primary syphilis unknown. "Unable to control legs" for nine years. Ataxic gait. Romberg's sign strongly positive. Pupils pin-point and equal; no reaction to light. Knee-jerks absent. Lower half of body almost completely anæsthetic. Wassermann reaction strongly positive (+ +).

URINARY SYMPTOMS.—Subnormal desire to micturate. No retention. No dribbling. No enuresis.

CYSTOSCOPY.—Residual urine 300 c.c. Urethra nearly anæsthetic. Bladder distended with 200 c.c. No cystitis. Slight trabeculation. No funnel-neck deformity. Bladder then filled to 1000 c.c. fluid; no discomfort. Well-marked funnel-neck deformity then observed (*Type IV*).

Case 10.—R. D. Aged 60. *Tabes dorsalis*.

Primary syphilis over thirty years ago. No immediate treatment. "Gastritis" nine years ago; at this time received nine months' treatment for syphilis. Diagnosed as *tabes dorsalis* ten months ago. Complaints of weakness of legs for past two months. Sciatica in thighs and calves for several years. Pupils; very sluggish reaction to light. Knee- and ankle-jerks absent. No areas of anæsthesia detected. Wassermann test (blood) positive. Has received approximately ten months' treatment for neurosyphilis.

URINARY SYMPTOMS.—Infrequent micturition and passage of large quantities of urine. Subnormal desire to micturate. F.=D.2-3/N.o. Good stream, but interrupted at times. Occasionally urine dribbles away into his clothes. Nocturnal enuresis on a few occasions. No retention.

CYSTOSCOPY.—Residual urine 120 c.c. Urethral sensation apparently much diminished. Bladder filled with 200 c.c. fluid. Fine trabeculation of bladder wall. No cystitis. Slight degree of funnel-neck urethra (*Type II*). Bladder filled to 400 c.c. Well-developed funnel-neck urethra (*Type IV*). When bladder was distended to 625 c.c. some mild discomfort was felt.

CYSTOGRAPHY.—Cystogram (500 c.c.) showed smooth regular bladder outline. Slight funnel-neck deformity.

Case 11.—W. M. Aged 61. *Tabes dorsalis*.

Twelve years ago, followed an alcoholic debauch, developed retention. Catheterized. No history of venereal disease. No evidence of stricture or prostatic enlargement. Knee-jerks normal at this time. Subsequently attended hospital at varying intervals complaining of retention of urine, but on examination bladder was never distended. Cystoscopy performed nine years ago. Bladder showed slight trabeculation and false diverticula; no cystitis. No observation was made on bladder neck. During past year patient's symptoms have been more pronounced. Attends hospital regularly complaining of retention, and stating that he has not passed urine for several days, but that a little dribbles away. On catheterization never more than 75 to 100 c.c. urine obtained. No. 24 F. bougie passed easily. Eight months ago sprained left ankle, and since then a typical Charcot's joint has developed. Wassermann and Kahn reactions strongly positive.

CYSTOSCOPY.—Urethral sensation slightly diminished. Bladder distended with 200 c.c. fluid. No marked cystitis. Fine isolated trabeculations and false diverticula. No evidence of funnel-neck deformity. Slight prostatic hypertrophy.

Case 12.—A. R. Aged 51. *Tabes dorsalis*.

Date of primary syphilis unknown. Diagnosed as locomotor ataxia six months ago. History of "weary feelings" and headaches for previous two years. Mind obviously clouded. Pin-point pupils; no reaction to light. Knee- and ankle-jerks absent. Sensation over lower half of body markedly impaired. Wassermann test strongly positive. Received six months' treatment for neurosyphilis prior to this investigation.

URINARY SYMPTOMS.—"Can hold water for a long period." Stream interrupted. Believes he has finished and then has to start again. Occasional dribbling. No nocturnal enuresis.

CYSTOSCOPY.—Urethral sensation very diminished or absent. Urine clear. Bladder filled with 200 c.c. fluid. No cystitis present. Funnel-neck urethra (*Type IV*).

Case 13.—F. F. Aged 43. *Tabes dorsalis*.

Primary syphilis twenty years ago. Complained of pains in legs twelve years ago. Wassermann test positive at that time. Received antisyphilitic treatment for two years. Attended a Municipal Clinic six years ago. Diagnosed as a case of *tabes dorsalis* at this time. Has received treatment for neurosyphilis ever since. Two years ago developed Charcot's disease of left hip. Left knee-joint also disorganized. Now shows marked Romberg's sign. Pupils equal but react very sluggishly to light. Knee- and ankle-jerks absent. Some loss of sensation over whole body below waist line; very marked loss from knees down and over saddle area.

URINARY SYMPTOMS.—Difficulty in micturition for five to six years. Never complete retention. Normal desire to micturate. $F.=D.3/N.O.$ Does not pass very large quantities. No dribbling. No enuresis.

CYSTOSCOPY.—Urine clear. Urethral sensation severely diminished. No residual urine. Bladder filled with 200 c.c. fluid. No cystitis. No trabeculation. Funnel-neck bladder (*Type II*). Bladder distended to 600 c.c.; slight discomfort. Funnel-neck more conspicuous (*Type IV*). Verumontanum well seen.

Case 14.—R. C. Aged 22. *Tabes dorsalis* (juvenile).

Congenital syphilis. Sought treatment three months ago on account of marked inco-ordination and difficulty in walking. Diagnosed as *tabes dorsalis*. Wassermann test (blood) positive. Cerebrospinal fluid showed greatly increased globulin; Wassermann test positive. Has now received three months' antisyphilitic treatment. His walking is stated to have improved. Tremors and ataxia of all limbs present. Scanning speech. Pupils contracted and fixed. Knee- and ankle-jerks absent.

URINARY SYMPTOMS.—Patient states that there is no incontinence, but hospital attendants report continual dribbling of urine. No retention. $F.=D.4/N.O.$

CYSTOSCOPY.—Urethra almost anæsthetic. Residual urine 480 c.c. Bladder filled with 200 c.c. fluid. Mucosa normal. No cystitis. No marked trabeculation. Funnel-neck bladder (*Type IV*). The colliculus was visible even though the prostatic urethra was not greatly dilated. Bladder distended to 800 c.c. fluid, at which point mild discomfort was felt. The prostatic urethra was still not widely dilated.

Case 15.—T. H. Aged 36. Tabes dorsalis.

Date of primary syphilis unknown. Lightning pains in his legs and arms for two years. Difficulty in walking in the dark. Pupils pin-point, no reaction to light. Knee-jerks very sluggish. No loss of sensation. Hyperæsthesia over arms and back. Wassermann test positive. Has received antisyphilitic treatment.

URINARY SYMPTOMS.—Difficulty in micturition. Believes desire to micturate is sub-normal. Strains to urinate, stream poor and interrupted. May pass only a trickle of urine or at other times one or two pints. Following urination bladder does not feel empty. Catheterizes himself. Frequency varies from once a week to twice daily. No dribbling. No nocturnal enuresis, but his bowels have occasionally moved during sleep.

CYSTOSCOPY.—No residual urine. Low urethral sensibility. Feels pull on ligaments. Bladder filled with 200 c.c. fluid. Cystitis, very slight. Trabeculation practically absent. No funnel-neck deformity. Distension increased to 350 c.c., but no change in appearance of bladder neck.

Case 16.—W. J. Aged 60. Tabes dorsalis.

Gonorrhœa thirty years ago. Developed a stricture six years ago and attended a genito-urinary clinic. Tabetic gait noticed at this time and diagnosed as tabes dorsalis. Wassermann test (blood) positive (+ +). Has since received continuous treatment for neurosyphilis. At present marked ataxia of lower limbs. Pupils small, very slight reaction to light. Knee-jerks present.

URINARY SYMPTOMS.—Absence of desire to micturate. Can urinate freely. Never retention. No dribbling. No enuresis. F.=D.3/N.o.

CYSTOSCOPY.—Urethra relatively anæsthetic. Residual urine 30 c.c. Bladder filled with 200 c.c. fluid. No cystitis. Beautiful fine trabeculation all over roof of bladder. Funnel-neck present (*Type IV*). The bladder was further distended, and when it contained 700 c.c. slight discomfort was felt. The dilatation of the prostatic urethra had to some extent increased. Excellent view of verumontanum and prostatic utricle.

Case 17.—J. O. Aged 46. Tabes dorsalis.

Date of primary syphilis unknown. Diagnosis of tabes dorsalis established one year ago. Argyll Robertson pupils. Knee- and ankle-jerks absent. Ataxia. Romberg's sign positive. Wassermann test (blood) positive. For past year has been receiving treatment for neurosyphilis.

URINARY SYMPTOMS.—Normal desire to micturate. Good stream. F.=D.4/N.o. No retention or difficulty. No dribbling. Enuresis on four or five occasions about twelve months ago. No trouble since.

CYSTOSCOPY.—No discomfort on insertion of cystoscope. Residual urine 450 cc. Bladder filled with 200 c.c. fluid. No cystitis. No abnormal degree of trabeculation. Mild degree of funnel-neck (*Type II*). Bladder distended to 500 c.c. without discomfort. Funnel-neck deformity developed considerably, the colliculus now being well seen (*Type IV*).

Case 18.—G. P. Aged 34. G.P.I. (early).

Primary syphilis 1922. Received treatment for one year. Epileptiform seizure six months ago; mentally confused for twenty-four to thirty-six hours, after which his mind made a complete recovery and he had no residual paralysis. The blood Wassermann at this time gave a doubtful positive reaction, the cerebrospinal fluid was strongly positive. He now complains of unsteadiness in the dark. Jaundice developed one month ago and has persisted. The pupils are not abnormally contracted, and react to light and accommodation. Knee- and ankle-jerks present. Sensation normal.

URINARY SYMPTOMS.—No retention. Slight dribbling of urine for two years, not sufficient to soil his clothes. F.=D.4/N.o.

CYSTOSCOPY.—Bladder filled with 200 c.c. fluid. The mucosa was tinged a distinct yellow (jaundice). Very little trabeculation—within normal range. On withdrawing the the cystoscope into the urethra the field appeared dark at first; on further withdrawal the colliculus was well seen in a brightly illuminated cavity (*Type III*). Bladder distended to 600 c.c. without discomfort. Fully developed funnel-neck (*Type IV*).

Case 19.—W. E. W. Age 40. G.P.I.

Primary syphilis twelve years ago. Received three injections at this time but no further

treatment. Two years ago became very nervous and developed marked tremors. Memory also became impaired. Later attended Salford Municipal Clinic, where a diagnosis of G.P.I. was made. Wassermann test (blood) positive. The cerebrospinal fluid contained 106 cells per c.mm.; globulin + + +; Wassermann and Kahn tests positive. Colloidal gold test showed a paretic curve. Has received a year's treatment for cerebrospinal syphilis, including malarial treatment six months ago. Pupils now unequal and give no reaction to light. Knee- and ankle-jerks all present and normal. Sensation appears normal, no loss in saddle area.

URINARY SYMPTOMS.—Normal desire to micturate. No difficulty; no straining. F.=D. hourly/N.o-I. No history of retention. Sexual intercourse normal, normal discharge.

CYSTOSCOPY.—480 c.c. urine withdrawn from bladder (represents two hours). Urine clear. Bladder filled with 200 c.c. fluid. Moderate degree of trabeculation of ordinary type, came and went. No cystitis. Funnel-neck (*Type III*) present. Verumontanum seen. Bladder distended to 400 c.c. without increasing degree of funnel-neck deformity.

Case 20.—V. C. H. S. Aged 50. G.P.I.

Contracted syphilis in Palestine during World War. Four years ago was diagnosed to be suffering from G.P.I. Shortly afterwards was admitted to an asylum suffering from delusions and paralysis of his right side. Wassermann test (blood and cerebrospinal fluid) positive (+ +). Received malarial treatment and made a good recovery. Discharged from asylum and commenced to attend Salford Municipal Clinic for treatment for neurosyphilis. At time of present examination has received two and a half years' treatment. Working for past two years and feels well. The pupils are small, the left reacts sluggishly to light, the right is fixed. The knee- and ankle-jerks are present. No anæsthesia demonstrated over legs or saddle area.

URINARY SYMPTOMS.—Normal desire to micturate. No dribbling. No enuresis. No retention. F.=D.4/N.o.

CYSTOSCOPY.—Urethra almost anæsthetic, no discomfort at cystoscopy. Residual urine 30 c.c. No cystitis present. No trabeculation seen. Bladder neck dilated (*Type II* or *IV*). It was possible to see the urethra extremely well for half to three-quarters of an inch. Though the verumontanum was then visible the surrounding space was inconsiderable. The bladder was distended to 800 c.c., when slight discomfort was felt. No change seen at bladder neck.

Case 21.—C. C. Aged 67. G.P.I.

Primary syphilis eighteen years ago. Attended urological clinic complaining of frequency, and incontinence of urine. No marked psychical changes. Face rather mask-like. Speech slurred. Pupils contracted, no reaction to light. Knee- and ankle-jerks increased. Slight spastic ataxia. No marked loss of sensation. Wassermann and Kahn tests (blood) positive (+ +).

URINARY SYMPTOMS.—Incontinence, clothes soaked with urine, apparently not a continuous dribble but active incontinence. F.=D.6/N. approx. every 20 min. Difficulty in voluntary micturition.

CYSTOSCOPY.—Urethral sensation not completely lost. Residual urine 525 c.c. (urine passed three-quarters of an hour previously). Bladder filled with 200 c.c. fluid. Mild injection of mucosa. Beautiful fine trabeculation all over sides and vault of bladder. Atrophic appearance. Slight degree of prostatic enlargement, small middle lobe present. Funnel-neck urethra (*Type IV*) very marked. Excellent view of colliculus. The degree of funnel-neck deformity was much too great to be accounted for by the minor degree of prostatic enlargement. Bladder distended to 600 c.c. No discomfort. Condition of bladder neck unchanged.

Case 22.—H. A. Aged 45. G.P.I.

No history of venereal disease. Attended hospital complaining of urinary incontinence. During past year has suffered from epileptiform seizures—two to four attacks weekly. Many facial cuts. Face mask-like. No marked psychical changes. Argyll Robertson pupil. Abdominal reflexes very brisk. Knee- and ankle-jerks increased. Extensor plantar response on right side. No definite loss of sensation. Wassermann test (blood) positive (+ +); Kahn test positive (+).

URINARY SYMPTOMS.—Incontinence; slight but continual dribbling of urine for past two years; wets his clothes; more severe at night; increased by active movement. No

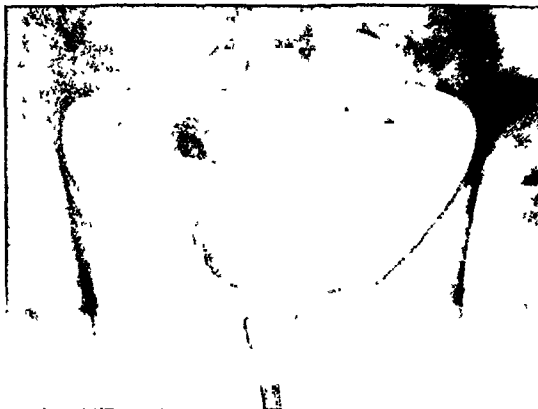


FIG. 364.—*Case 22.* G.P.I. Cystogram showing bladder filled with 200 c.c. potassium iodide solution. Resting pressure 30 c.c. of water. Cystogram made whilst viewing the deformity through the cystoscope.



FIG. 365.—*Case 22.* G.P.I. Cystogram showing condition as in previous figure but with cystoscope removed.



FIG. 366.—*Case 22.* G.P.I. Cystogram following partial emptying of the bladder by voluntary effort, showing the deformity still present.

active incontinence. Voluntary micturition difficult; may wait ten minutes for act to commence; straining; doubles himself up; interrupted stream. $F.=D.3/N.1-o$. Impotent.

CYSTOSCOPY.—Little diminution in urethral sensation. Residual urine 420 c.c. Urine clear. Bladder filled with 200 c.c. fluid. No cystitis. Well-marked fine and coarse trabeculation, false diverticula. Very pronounced funnel-neck deformity (*Type IV*). Trigone merges almost imperceptibly into posterior urethra. Magnificent view of verumontanum.



FIG. 367.—Case 22. G.P.I. Excretion cystogram. There is no sign of a funnel-neck deformity.

CYSTOGRAPHY.—Solution introduced with cystoscope in position (200 c.c.; resting pressure 30 cm. water). Cystogram, taken whilst observing deformity through cystoscope, showed a large funnel-neck deformity (*Fig. 364*). Following removal of cystoscope further cystograms still showed the deformity (*Fig. 365*). An attempt was made to introduce 200 c.c. more fluid through a catheter, but the patient complained of severe irritation in the bladder, and was unable to retain the fluid. A final cystogram with a very small quantity of fluid in the bladder still showed the deformity (*Fig. 366*). A subsequent excretion cystogram, however, showed no sign of funnel-neck (*Fig. 367*).

Case 23.—J. W. G. Aged 37. Cerebrospinal syphilis (Erb's syphilitic spinal paralysis).

Date of primary syphilis unknown. Ten months ago commenced to suffer from spastic weakness in his right foot. A month or two later both legs were involved and marked urinary symptoms were present. Diagnosed as Erb's syphilitic spinal paralysis five months ago. Spastic gait. Pupils give sluggish reaction to light. Knee- and ankle-jerks unusually active. Ankle- and patella-clonus present. Extensor plantar response on stimulation. Some anæsthesia over lower limbs but not well marked. Wassermann and Kahn tests positive (+ +). Received five months' treatment for neurosyphilis prior to examination.

URINARY SYMPTOMS.—Incontinent for past eight months. Urine does not dribble away; suddenly micturates and soaks his clothes (active incontinence). Nocturnal enuresis. Does not feel normal impulse to micturate. No retention. $F.=D.3-4/N.1$ (and several times into his clothes).

CYSTOSCOPY.—Urethral sensation very low. Residual urine 420 c.c. Bladder filled with 200 c.c. fluid. No cystitis present. Well-marked and very pretty trabeculation of bladder. Funnel-neck (*Type IV*). Colliculus well seen.

Case 24.—W. J. M. Aged 48. Cerebrospinal syphilis.

Gonorrhœa twenty years ago. Attended urological clinic one year ago on account of diurnal and nocturnal incontinence of urine and also marked frequency of micturition. Sudden onset of symptoms. Bladder distended and palpable for approximately 1½ in. above the pubis. Patient also complained of difficulty in walking for the previous fifteen months.

Toes tended to catch the ground. No lightning pains. Pupils normal. Knee-jerks present (+ +). Wassermann reaction positive (+ +).

A cystoscopic examination was performed at this time. The bladder was distended to 200 c.c. The mucosa was hyperæmic and showed marked trabeculation. The posterior urethra was deepened and dilated. The colliculus was enlarged and easily visible (*Type IV*).

The patient has now received antisyphilitic treatment for one year.

Cystoscopy.—Residual urine, 480 c.c. No cystitis. Trabeculation very marked. Funnel-neck (*Type III*). Definite waist intervening between the bladder and the dilated posterior urethra. Colliculus well seen.

II. OBSERVATIONS ON THE NEUROLOGICALLY HEALTHY MALE

This work has been carried out on patients attending the Cystoscopic Clinic for routine examination. All those who were known to be suffering from nervous disease, or who subsequently developed signs of such, have been excluded. Cases of prostatic hypertrophy have also been rejected. The results have been divided into two series (*Table II*). Series I (87 cases) represents a period when no special routine observation for the deformity was kept on the vesical orifice, and the phenomenon was noted in only 3.5 per cent of cases. In Series II (240 cases) a routine examination was made in every patient, and a funnel-neck deformity (*Type III* or *IV*) was found in 7.5 per cent.

Table II.—CYSTOSCOPY IN THE NEUROLOGICALLY HEALTHY MALE.

	SERIES I	SERIES II
Number of males	87	240
Cases of funnel-neck deformity	3	18
Percentage	3.5	7.5

In *Table III* (p. 526) details of those patients exhibiting a positive phenomenon are recorded. Unfortunately, as so frequently happens in clinical investigation, absolutely complete data have not been obtained in each case. Thus when a funnel-neck deformity has been detected under local or sacral anæsthesia, it has not always been possible to confirm its presence on a subsequent cystoscopy.

Weakness of the internal sphincter in the absence of nervous disease requires explanation. Fatigue and straining to micturate are two possible causative factors, but every endeavour has been made to exclude them.

It is our routine practice to use either local anæsthesia (1-1000 percaine) or, more rarely, spinal or sacral anæsthesia, and therefore all patients have had one or other of these anæsthetics. Of the 21 positive cases, 12 exhibited the phenomenon following either spinal or sacral anæsthesia. The number of patients to whom such anæsthetics are given is small, and therefore the large number exhibiting the phenomenon suggests that this form of anæsthesia is of etiological importance. The fact that in three cases where a confirmatory cystoscopy was carried out the sign was absent, also supports this view (*Cases 4, 13, 17*). In *Case 4* the urethral orifice was rather patulous, but this was probably accounted for by the fact that a small papilloma at the urethral orifice had been destroyed by diathermy, with possible partial destruction of the internal sphincter. In two instances (*Cases 3 and 9*) the deformity was present in the absence of an anæsthetic.

To the remaining 9 cases local anæsthetics were given. The presence of the deformity in the absence of the anæsthetic was confirmed in three instances (*Cases 11, 18, and 20*) (*Fig. 368*). In a fourth case the regurgitation of fluid up the left ureter on a cystogram suggested some lack of tonicity in the bladder wall. In *Case 11* the Wassermann reaction is positive, and although there are no neurological signs the patient is commencing to suffer from slight incontinence and is probably an early tabetic. These cases suggest that local anæsthesia may cause

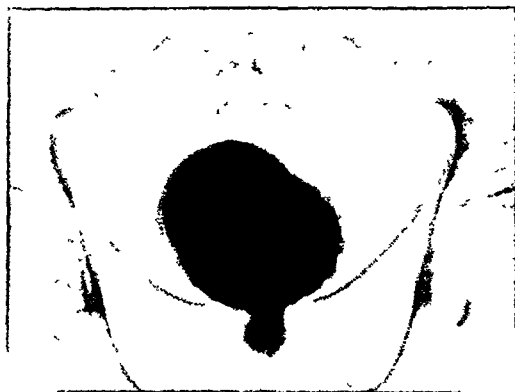


FIG. 368.—*Case 18*. Cystogram from a non-neurological patient showing funnel-neck deformity.

funnel-neck deformity, but that it is of less importance than sacral or spinal anæsthesia in this respect. A pronounced degree when encountered under local anæsthesia points to the advisability of further cystoscopic and neurological examination.

Excluding all doubtful and unconfirmed cases there remain four instances of funnel-neck deformity (*Cases 3, 9, 18, and 20*) in which no cause for the lesion can be demonstrated. It is thus probable that the sign may appear from time to time in the neurologically healthy male, and that it is not definitely pathognomonic of nervous disease.

CONCLUSIONS

1. Funnel-neck deformity of the bladder is very frequently observed in syphilitic disease of the central nervous system. The work of other writers shows that it is not confined to this complaint. The phenomenon is due to paresis of the internal vesical sphincter.

2. Dilatation of the internal vesical sphincter occurs during micturition, and thus minor and transient degrees of the phenomenon may be observed during straining at cystoscopy. Spinal, sacral, or local anæsthesia may produce varying degrees of the condition. The sign is therefore not absolutely pathognomonic of nervous disease, and is only of diagnostic significance when these factors are absent.

3. The deformity may rarely occur in the apparently healthy male.

4. Although visible on cystoscopic examination, cystography may fail to demonstrate the sign. This is due either to sufficient tone remaining in the internal sphincter to close the bladder neck, or to the elasticity of the tissues in this region.

Table III.—NON-NEUROLOGICAL CASES SHOWING FUNNEL-NECK DEFORMITY

No.	Ref.	AGE	DISEASE	W.R.	ANÆSTHETIC	CYSTOSCOPY: DEGREE OF DEFORMITY	CONFIRMATORY CYSTOSCOPY WITHOUT ANÆSTHETIC	CYSTOGRAM OR EXCRETION UROGRAPHY
1	O. T. . .	32	Frequency and dysuria	Neg.	Sacral and evipan	Type IV	Resists any examina- tion unless under general anæsthetic	—
2	W. R. . .	50	Urological examination for hæmaturia	Neg.	Sacral . .	Type IV	—	—
3	A. N. . .	17	Dysuria	Neg.	1. Sacral . . 2. Spinal . .	Type IV	—	Excretion urogram shows funnel-neck deformity
4	J. W. . .	49	Papilloma of bladder	Neg.	1. Sacral . . 2. Sacral . .	Type IV Type IV	Patulous urethral ori- fice (Type II)	—
5	T. B. . .	31	L. hydronephrosis . .	Neg.	1. Local . . 2. Local . .	Type III Type III or IV	No funnel-neck. On straining Type III developed	—
6	B. M. . .	50	L. ureteric calculus . .	—	Sacral . .	Type III	—	—
7	S. W. . .	56	Chronic interstitial ne- phritis	—	Sacral . .	Type III or IV	—	Excretion urogram shows no abnormality
8	S. H. . .	52	Testicular pain. Fre- quency	—	Sacral . .	Type IV	—	—
9	J. L. . .	54	Vesico-colic fistula . .	Neg.	1. Local . . 2. Spinal . .	Type IV Type IV	Funnel-neck deform- ity (Type IV) present	—
10	G. C. . .	38	L. renal colic . .	—	Sacral . .	Type IV	—	Excretion urogram showed no funnel- neck deformity

II	C. D. ..	44	Papilloma of bladder ..	Pos. (+ +)	1. Local 2. Local	Type IV Type IV	Funnel-neck deform- ity (Type IV)	Cystogram: marked trabeculation; slight funnel-neck deform- ity
12	H. F. ..	37	Chronic prostatitis ..	—	Sacral	Type IV	—	—
13	E. B. C.	59	Papilloma of bladder ..	Neg.	1. Local 2. Sacral 3. Sacral	No funnel-neck noted. Verumontanum just visible. Type IV	No funnel-neck de- formity. On strain- ing Type IV de- veloped	—
14	G. A. G.	36	Hemospermia ..	Neg.	Local	Type IV	—	No trabeculation. No funnel-neck
15	W. M. ..	26	Renal investigation for L. lumbar pain	Neg.	Local	Type IV	—	Slight trabeculation. No other deformity
16	H. W. F.	38	L. pyelonephritis ..	—	Local	Type IV	—	—
17	N. R. ..	36	Papilloma of bladder ..	—	1. Local 2. Sacral	No funnel-neck de- formity Type IV	No funnel-neck de- formity	—
18	J. B. ..	35	R. renal calculus ..	Neg.	Local	Type IV. Increased with straining	Funnel-neck deform- ity (Type IV). In- creased a little on straining	Funnel-neck deform- ity (Fig. 368). Slight trabeculation
19	O. P. ..	26	Renal investigation for R. ureteric calculus	—	Local	Type IV	—	—
20	K. L. ..	12	Nocturnal enuresis ..	—	Local	Type IV	—	Cystogram: no trabeculation; small funnel-neck
21	F. R. ..	26	Glandular hypospadias. Frequency	Neg.	Local	Type IV	—	Cystogram: no trabeculation; no funnel-neck; regur- gitation up L. ureter

5. Ascending cystography is not a satisfactory method of examining the vesical neck. Excretion cystography offers considerable advantages.

In conclusion, the authors sincerely thank Dr. E. Tytler Burke, Dr. J. Dudgeon Giles, and Dr. W. A. Ramsay for their great help in providing material for this investigation.

BIBLIOGRAPHY

- ALEXEIEFF, N., "Protokoll der Sitzung der urologischen Gesellschaft zu St. Petersburg am Feb. 19, 1914", quoted by Epstein; *Zeits. f. Urol.*, 1914, quoted by Natanson and Epstein.
- BARNEY, J. D., *Boston Med. and Surg. Jour.*, 1910, clxiii, 933, 985.
- BARRINGTON, F. J. F., *Quart. Jour. Exper. Physiol.*, 1914, viii, 33.
- BÖHME, *Münch. med. Woch.* 1908, lv, 2610.
- BRAASCH, W. F., *Trans. Amer. Med. Assoc.* (Sect. on Genito-Urinary Diseases), 1917, 48.
- BRAASCH and HAGER, *Surg. Gynecol. and Obst.*, 1927, xlv, 442.
- BURNS, E. J., *Ibid.*, 1917, xxiv, 659.
- CAULK, J. R., and GREITZER, H. G., *Interstate Med. Jour.*, 1916, xxiii, 36.
- CAULK, J. R., GREITZER, H. G., and BARNES, F. M., *Jour. Amer. Med. Assoc.*, 1919, lxiii, 1594.
- CUMMING, R. E., *Jour. Amer. Med. Assoc.*, 1922, lxxviii, 335; *Ibid.*, 1932, xcix, 1998.
- DEUTSCH, I., *Zeits. f. Urol.* 1926, xx, 1.
- DENNY-BROWN, D., and GRAEME ROBERTSON, E., *Brain*, 1933, lvi, 397.
- DUNAJEWSKY and MICHEJEW, W. W., *Zeits. f. urol. Chir.*, 1931, xxxiii, 334.
- EPSTEIN, G. S., JUSCHELEVSKY, A. S., and MICHELSON, J. D., *Zeits. f. Urol.*, 1929, xxiii, 35.
- FESSLER, L., and FUCHS, F., *Zeits. f. urol. Chir.*, 1929, xxxviii, 175.
- FRANKL-HOCHWORT, *Assoc. Internat. d'Urol.*, 1908, i, quoted by Barney.
- GAGSTATTER, *Verhandl. d. Wien. urol. Gesells.*, 1923, quoted by O. A. Schwarz.
- GÁSPÁR, F., *Zeits. f. urol. Chir.*, 1935, xli, 117.
- GOLDENBERG, T., *Verhandl. d. deut. urol. Gesells.*, 1924.
- GOWERS, W. R., *Diseases of the Nervous System*, 1886, i, 299. London.
- GREENBERG, G., *Med. Record*, 1916, xc, 634.
- GUTHRIE, G. J., *Anatomy and Disorders of the Neck of the Bladder*, 1834, 15. London.
- HALPERSTEIN, *Karzan. med. Z.*, 1928, No. 6, quoted by Dunajewsky.
- HEAD, H., and RIDDOCH, G., *Brain*, 1917, xl, 188.
- HEPLER, A. B., *Surg. Clin. N. Amer.*, 1933, xiii, 1379.
- HOLMES, G., *Brain*, 1933, lvi, 383.
- KOLL, I. S., *Surg. Gynecol. and Obst.*, 1915, xx, 176.
- LEARMONTH, J. R., *Brain*, 1934, xlv, 147.
- MACALPINE, J. B., *Proc. Roy. Soc. Med.* (Urol. Sect.), 1934, xxviii, 1.
- MCCREA, E. D., *Irish Jour. Med. Sci.*, 1927, 658.
- MEYERS, I. L., *Jour. Nerv. and Ment. Dis.* 1926, lxiv, 321.
- MORO, N., *Zeits. f. Urol.*, 1927, xxi, 424.
- NATANSON, H., *Ibid.*, 821.
- NITZE, M., *Lehrbuch der Kystoskopie* (2nd ed.), 1907, 260.
- PERLMANN, S., *Zeits. f. Urol.*, 1926, xx, 501.
- PISTER, E., *Ibid.*, 1921, xv, 343.
- PLAGEMEYER, H. W., *Jour. of Urol.* 1919, iii, 367.
- RANDALL, A., *New York Med. Jour.*, 1915, cii, 1182.
- SCHRAMM, K., *Zeits. f. Urol.*, 1920, xiv, 329; *Verhandl. d. deut. urol. Gesells.*, 1926, quoted by Natanson.
- SCHWARZ, OSWALD, *Lichtenberg, Voelcker, and Wildbolz's Handbuch der Urologie*, 1926, i, 459.
- SCHWARZ, O. A., *Zeits. f. Urol.*, 1925, xix, 350.
- SCHWARZWALD, *Verhandl. d. Wien. urol. Gesells.*, quoted by O. A. Schwarz.
- THOMSON-WALKER, J. W., *Ann. of Surg.*, 1910, lii, 577.
- VON DER BECKE, A., *Rev. Assoc. méd. argent.*, 1932, xlv, 1302.
- WALKER, KENNETH, *Report of the 5th Congress of the International Society of Urologie*, 1926, i, 516. London.
- WATKINS, K. H., *Brit. Jour. Surg.*, 1936, xxiii, 734.
- WEIJTLANDT, J. A., *Report of the 5th Congress of the International Society of Urology*, 1933, i. London.

THE ASSOCIATION OF INGUINAL HERNIA WITH TRAUMATIC PERFORATION OF THE INTESTINE*

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THE object of this communication is to illustrate the relation which inguinal hernia bears to traumatic perforation of the intestine, as the result of direct injury to the hernia or abdominal wall, or indirectly as the result of excessive muscular effort. The association appears to be definite, but only a few references to it occur in the literature, and these chiefly in German.

CASE REPORTS

Case 1.—An insurance agent, aged 51, was admitted on the morning of June 25, 1921, to the Royal Infirmary, Edinburgh. A bilateral inguinal hernia had been present for ten years. On the previous afternoon, in a race at a picnic, the patient had tripped and had fallen heavily upon the abdomen. Although severely winded, he had suffered no immediate great pain, but half an hour later an increasing colic had developed below the umbilicus. During the night the pain had become unbearable, and had spread upwards into the epigastrium. There had been no vomiting, and at no time had there been symptoms referable to either hernia. The temperature was 100.2°, the pulse 90, and the respirations 20 per minute. The skin was tense over a distended abdomen. Both herniæ were reducible. Hyperæsthesia and marked tenderness were elicited in both iliac fossæ, and a general abdominal rigidity was greatest below the umbilicus. There was dullness in both flanks.

At operation, under general anæsthesia, the peritoneal cavity was opened by a right paramedian incision, and found to contain purulent fluid. The dilated coils of intestine were clothed in fibrinous exudate. On separation of two adherent coils of ileum a perforation was found at the mesenteric border of the bowel. This perforation had apparently occurred initially between the leaves of the mesentery, which had ruptured later to permit general peritoneal infection. A clamp having been applied to the base of the perforated loop, the perforation was closed and invaginated, and the mesentery repaired. The peritoneal cavity was drained through a transverse suprapubic incision. Progress after operation was at first satisfactory, but on the third day hiccup developed, and vomiting, at first of bile and later fæcalent fluid, became almost continuous. Death occurred in pyrexia on the fourth day. Post-mortem examination showed a considerable degree of purulent peritonitis, and a general intestinal dilatation.

Case 2.—A greenkeeper on a golf course, aged 71, was carried to the Royal Infirmary immediately after a road accident. Walking home in the roadway under the influence of alcohol, he turned at the sound of a motor-car behind him, and was struck by the car, probably in the belly. He was thrown clear, and rose at once, partly stunned and complaining of severe abdominal pain. There were no striking signs of shock. The pulse was full, strong, and regular, with a rate of 96 per minute. The temperature was 97.4°, the respirations 24 and abdomino-thoracic. The face and nose were bruised. Cerebration was slow and speech incoherent, largely from the effects of alcohol. The abdomen presented no external signs of violence. A reducible right inguinal hernia extended down into the scrotum. The abdomen moved freely with respiration. There was slight tenderness two inches below and to the left of the umbilicus, but no appreciable rigidity. On examination two hours later the abdominal wall was generally rigid, most markedly so to the left of the midline, and tenderness also was generalized.

At operation under general anæsthesia, through a left pararectal sub-umbilical incision, the abdomen was opened and the peritoneal cavity found to contain a small quantity of clear fluid. Tiny subserous hæmatomata were present, scattered over various loops of bowel.

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A loop of small intestine was withdrawn and the gut followed downwards to a perforation, large enough to admit a finger-tip, at the lower end of the ileum. Little or no dissemination of intestinal contents had occurred. Resection of the injured loop was performed, with end-to-end reconstruction. The abdomen was closed and suprapubic drainage of the peritoneal cavity established. Rapid recovery was interrupted by mild infection of the abdominal wound.

Case 3.—A grocer's assistant, aged 52, was admitted to the Royal Infirmary on May 23, 1934. A few hours before admission he had slipped in descending from a tramcar, and had saved himself from falling only by a sudden violent hyperextension of the trunk. He experienced a severe pain simultaneously in the epigastrium and in a right inguinal hernia which had been present, reducible, for years. The pain abated a little, but returned with suddenly renewed severity an hour later, to spread from the epigastrium rapidly over the abdomen, and to persist until admission to hospital. Vomiting occurred once. Increasing indigestion, present for years, and taking the form of epigastric pain an hour after food, had culminated in a recent hæmatemesis, treated in hospital. The temperature was 98°, the pulse-rate 90. The patient, slightly cyanosed, and groaning from pain, lay motionless on his back. Respiration was shallow and entirely thoracic. The abdomen was slightly distended, uniformly and absolutely rigid, and generally tender. Tenderness was maximal in the epigastrium, and over a right inguinal enterocele, even after reduction of the contents of the hernia sac. Both flanks were dull to percussion, and the liver dullness was lost. The case was regarded as a typical one of perforated peptic ulcer. The history of injury was considered coincidental, and the hernial tenderness ascribed to the share taken by the hernia sac in the suspected general peritonitis.

Under general anaesthesia, through a right paramedian incision, the peritoneal cavity was opened and a large quantity of bile-stained fluid evacuated by suction. A fibrinous peritonitis was present. No perforation or ulceration was found in stomach or duodenum. Seven inches below the duodeno-jejunal junction was found a transverse tear of the gut, involving the anti-mesenteric border and two-thirds of the circumference. Through the tear the mucous membrane was pouting, gripped by the torn edges of the muscle layer. The perforation, which had occurred through healthy bowel wall, was closed in two layers, and the wound sutured after passage of a suprapubic drain into the rectovesical pouch. Recovery was uninterrupted.

Case 4 (by permission of Mr. I. H. McClure).—A ship's engineer, aged 56, while returning to his ship one evening, felt sudden severe pain over the symphysis pubis, where the pad of a truss inadequately and only partially controlled a long-standing right inguinal hernia. This pain increased, and rose gradually higher in the abdomen. Vomiting occurred and was repeated. When seen by Mr. McClure four hours later, the patient persisted in blaming his hernia for the attack of pain. It was obvious, however, that an early general peritonitis was present, and laparotomy was performed. A large quantity of turbid fluid was evacuated from the peritoneal cavity. The peritoneum was everywhere congested, and numerous petechial hæmorrhages were present. The lower ileum was considerably dilated, and a circular perforation was found, 4 mm. in diameter, in the antimesenteric border of the bowel a few inches above the ileocaecal valve. The perforation was 'punched-out', and there was no pouting of the mucosa. The perforation was closed and a temporary ileostomy performed above the closure. The patient was discharged three weeks later after an uneventful recovery.

DISCUSSION

Rupture of the intestine as a complication of ill-advised attempts to reduce an incarcerated inguinal hernia, or from direct trauma to the hernia, or even from the pressure of a truss, is not excessively uncommon, and over a hundred cases of general peritonitis from this cause have been reported, chiefly during the last century.^{4, 5, 12, 19, 20} Much less common is the variety of rupture of the bowel which, after abdominal violence or sudden muscular strain, may occur as a complication of inguinal hernia. It is this rare injury with which we are specially concerned; our first three cases bring to 33 the total number of reports of its incidence. While it is impossible to estimate the frequency of the condition, it is probably commoner than this figure suggests. Tschistosserdoff²⁹ observed that in

4 of his 52 cases of ruptured intestine a hernia was present, and Rubritius²¹ had 1 instance of the co-incidence in 13 injuries of the intestine. Taylor,²⁸ in 4 cases of perforation of the intestine, records in 3 the presence of a hernia, but fails to mention its possible importance.

It is interesting to note that the condition has not been observed in women; all the reported cases are in well-built, strong, and healthy men. Traumatic perforation of the intestine has never been recorded in a case of femoral hernia, or in an external hernia other than inguinal. The hernia has usually been old-standing, and usually, but not invariably, right-sided. The perforation may follow direct injury to the abdomen (as in our first two cases, and in most of the reported cases); it may result from sudden violent muscular strain (as in *Case 3* above).

The symptoms are those of a rapid, spreading, perforative peritonitis; localization by adhesions is rare. Frequently, after the initial pain, there is a latent interval in which the signs and symptoms of peritonitis may be almost entirely absent. The hernia is reducible in the majority of cases, and in all four cases reported above. There may or may not be special tenderness over the hernial swelling.

With the exception of Scarpa's case²³ (in which the colon was injured) the damage has been confined to some part of the small intestine. Although most common in the lower ileum, rupture may occur at any level. In most cases the perforation is situated at or close to the antimesenteric border; in only two instances (Campbell's⁴ and the first case above) has it occurred at the mesenteric attachment. The perforation may be either longitudinal or transverse, and the neighbouring bowel may present a bruised appearance, or a split in its serous coat. In most cases, the mucosa pouts slightly outwards, and is gripped by the edges of the opening in the muscle coat.

Except in one case in which the colon was injured within the sac,⁴ the perforated loop of intestine has invariably been found within the abdominal cavity. For this reason it is obvious that laparotomy rather than herniotomy is the treatment of choice. Herniotomy is favoured by certain German authors,^{7, 21, 27} and its frequent success is due mainly to the proximity, in most cases, of the injured loop to the abdominal inguinal ring. Usually the perforation is sufficiently small to permit closure and invagination by seromuscular suture, but occasionally an almost circumferential rupture demands resection of the damaged loop. In this, as in all traumatic ruptures of the small intestine, the mortality is high—in the neighbourhood of 66 per cent.

The mechanism of rupture of the intestine by direct or indirect violence in cases of inguinal hernia is not always obvious, and it is unlikely that a single explanation can be applied to all cases.

1. Where the causative violence has been applied directly to the hernia (as in our fourth case), the explanation is simple. The bowel is ruptured as it lies unprotected in the hernia sac.

2. It is more difficult to explain why the presence of a hernia should render the intestine more susceptible to injury by a force applied to a point on the abdominal wall remote from the hernia. It can be presumed that a fixed loop of bowel is more liable to injury than is a freely movable loop which can be dislodged in safety by a force applied to the abdomen.¹³ The first two cases recorded above immediately suggest that in these a loop of lower ileum, fixed on the one hand at the ileocaecal junction, fixed on the other at the abdominal inguinal ring by a

contraction of the abdominal muscles around the neck of the hernial sac, is drawn relatively taut. It may be broken across by the force applied to the abdomen, or, fixed in this way, it may be crushed between the external force and the posterior abdominal wall. The numerous bruises which presented on the serous surface of the injured bowel loop in our second case are strong proof that in that case, at least, some such mechanism was responsible. In our second case, the site of the tear at the mesenteric border suggests that the point of the abdominal fixation of the affected loop has been its own mesenteric attachment, a possibility which Kahn¹¹ has previously suggested.

3. Ruptures of the intestine resulting from violent hyperextension of the trunk, without direct injury to the abdomen, are still less easily explained. To account for these cases (which are well exemplified by the third case reported above), Bunge has advanced an ingenious hypothesis. He points out that any sudden rise of intra-abdominal pressure is invariably associated with a coincident rise in intra-intestinal pressure. In the normal abdomen such a rise of intra-intestinal pressure is of little consequence, since the pressure within the bowel is equal to the pressure around it at every point on its surface, and no rupture can occur. If, however, at the time of sudden elevation of intra-abdominal pressure a loop of bowel overlies the mouth of a hernia sac, and a rise of pressure consequently occurs in that bowel loop, the loop lacks the support of the intra-abdominal pressure at a localized point on its surface, protrudes downwards into the sac in which no rise of pressure has occurred, and bursts at that unsupported point. When the intra-abdominal pressure falls again, the ruptured loop moves away from the abdominal ring and discharges its content through the perforation to give a general peritonitis. A neat illustration of this mechanism is afforded by an inflated balloon placed in the barrel of a syringe. Provided the nozzle of the syringe be closed by the finger the balloon will fail to burst, however hard the piston is compressed upon it. As soon as the finger is removed from the nozzle the balloon protrudes through the nozzle where it is unsupported and bursts at that unsupported point. Bunge applied this explanation to all cases of hernia complicated by rupture of the intestine, whether by direct or by muscular violence, but the theory has its greatest value in explaining the rare cases in which the rupture occurs at a time when the hernial protrusion is not present.

Ingenious as the theory is, its complexity is a bar to its unqualified acceptance. The causative muscular violence is almost invariably a violent hyperextension of the trunk. It would be simpler to explain these cases by overstretching of a loop of bowel, fixed at one end at the ileocaecal or duodenojejunal junction, and gripped at the other by a contraction of the abdominal muscles around the hernial orifice. In our second case the perforation was seven inches below the ligament of Treitz. Bunge supports his theory by describing such injuries to the intestine as 'ruptures by bursting', but the direction of a perforation of the bowel and protrusion of the mucosa through a perforation are no certain indication of the mechanism of rupture. There is nothing in the descriptions of reported perforations of the small intestine in cases of hernia to prevent them being considered as 'ruptures by tearing'.

In conclusion, a loop of intestine lying within a hernia sac may be injured by violence applied directly to the hernial swelling—the pressure of an ill-adjusted truss, or too vigorous attempts to relieve an incarceration. Ruptures of the bowel caused by violence to the abdomen appear to be due to the application of the traumatic force to a doubly fixed and immovable bowel loop. Ruptures by

muscular violence are most simply explained by the overstretching of a bowel loop between the ileocæcal (or duodenojejunal) junction and the abdominal inguinal ring, and distraction of these points of fixation from each other by violent hyperextension of the trunk. Bunge's attractive theory of a bowel loop bursting outwards through the neck of a hernia sac (where the support offered by the intra-abdominal pressure is lacking) need be applied only to the excessively rare cases where the hernia sac, though unquestionably present, is empty at the moment of injury.

SUMMARY

In cases of abdominal injury followed by the clinical features of peritonitis, where an inguinal hernia is present, a rupture of the intestine should be suspected.

This rupture may be due to violence applied to the hernia itself, to direct abdominal injury, or, more rarely, to violent hyperextension of the trunk.

The mechanism responsible for the rupture of the bowel varies with the nature of the causative violence.

The first two cases of this series were patients of Mr. J. M. Graham, and the third case was also treated in his wards in the Royal Infirmary, Edinburgh. The author is grateful for permission to publish these cases, for access to their case-reports, and for the advice Mr. Graham has given in the preparation of the paper. The fourth case is published by permission of Mr. Ian H. McClure, of Kirkwall, Orkney, who has been generous also in providing a complete record of it.

REFERENCES

- ¹ BLINDOW, *Dissert.*, Berlin, 1885.
- ² BREUNING, W., *Zentralb. f. Chir.*, 1929, lvi, 726.
- ³ BROFELDT, S. A., *Acta chir. Scand.*, 1925, lvi, 256.
- ⁴ CAMPBELL, P., *Ann. of Surg.*, 1905, xlii, 661.
- ⁵ COOPER, A., *Surgical Essays*, 1833, 275.
- ⁶ GANGL, *Arch. f. klin. Chir.*, 1910, xciii, 292.
- ⁷ GRASSMANN, M., *Deut. Zeits. f. Chir.*, 1908, xci, 41.
- ⁸ HAIM, *Arch. f. klin. Chir.*, 1910, xciii, 685.
- ⁹ HILGENFELDT, O., *Ibid.*, 1931, clxvi, 219.
- ¹⁰ JUSTI, *Deut. Zeits. f. Chir.*, 1882, xvii, 172.
- ¹¹ KAHN, M., *Jour. Amer. Med. Assoc.*, 1914, lxi, 756.
- ¹² KAY, C. A., *Guy's Hosp. Rep.*, 1842, vii, 261.
- ¹³ LOCKWOOD, *Jour. Canad. Med. Assoc.*, 1923, ciii, 311.
- ¹⁴ LONGUET, *Bull. Soc. anat. de Paris*, 1875, 799.
- ¹⁵ VON MICKULICZ, *Sammlung. klin. Vorträge von Volkmann*, 262.
- ¹⁶ VON MORITZ, *Petersburg med. Woch.*, 1879, 457.
- ¹⁷ NEUMANN, *Deut. Zeits. f. Chir.*, 1902, lxi, 158.
- ¹⁸ OBERST, *Zentralb. f. Chir.*, 1885, xii, 345.
- ¹⁹ PETRY, *Beitr. z. klin. Chir.*, 1896, xvi, 545.
- ²⁰ POLAND, *Guy's Hosp. Rep.*, 1858, iv, 123.
- ²¹ RUBRITIUS, *Prag. med. Woch.*, 1905, 375, 390, 406.
- ²² SAUERBRUCH, *Mittel. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, xii, 93.
- ²³ SCARPA, *Traité pratique des Hernies*, Paris, 1823, 310.
- ²⁴ SCHÖNLEBER, W., *Beitr. z. klin. Chir.*, 1920, cxxi, 597.
- ²⁵ SEIGMUND, E., *Zentralb. f. Chir.*, 1928, lv, 1105.
- ²⁶ SOCIN, *Jahresberichte über die chirurg. Abteilung des Spitals zu Basel*, 403.
- ²⁷ TALKE, L., *Beitr. z. klin. Chir.*, 1907, lvi, 332.
- ²⁸ TAYLOR, J., *Edin. Med. Jour.*, 1925, xxxii.
- ²⁹ TSCHISTOSSERDOFF, W., *Beitr. z. klin. Chir.*, 1912, lxxiv, 70.
- ³⁰ VOSWINCKEL, *Arch. f. klin. Chir.*, 1906, lxxix, 505.
- ³¹ WIEDHOPF, *Zentralb. f. Chir.*, 1929, lvi, 39.
- ³² WINSLOW, R., *Trans. South. Surg. Assoc.*, 1918, xxx, 390.

CHRONIC PEPTIC ULCER OF THE ŒSOPHAGUS : A REPORT OF EIGHT CASES

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VARIOUS kinds of ulceration are found in the œsophagus. In this paper I do not propose discussing the malignant, syphilitic, or tuberculous ulcers, or even the so-called decubitus ulcer of the post-cricoid region, but shall confine myself to the simple chronic ulceration found usually in the lower end of the organ and frequently known as chronic peptic ulcer of the œsophagus.

Opinions as to the frequency of this interesting condition differ rather widely. Tileston,¹ in 1906, was able to collect only 8 cases from the literature. Stewart² stated that he had found only 1 case in a series of 10,000 post-mortems performed over a period of eighteen years. Stewart and Hurst,³ in their handbook on peptic ulceration, added 11 more cases to Tileston's 8. Chevalier Jackson,⁴ in 1929, by the routine use of the œsophagoscope in all cases of slight dysphagia, found 21 active ulcers and the scars of 67 in a series of 4000 cases. Excellent reproductions of the œsophagoscopic appearances of certain of them are given in his paper. His work has since been criticized by other writers, who have concluded that many of the ulcers described were probably acute in type. Friedenwald, Feldman, and Zinn,⁵ in 1929, came to the same conclusions as Jackson about the relative frequency of the condition, and published 13 cases from their own practice in which the condition was diagnosed by œsophagoscopy and X-ray examination. Eusterman, Moersch, and Camp,⁶ in 1930, reported 3 cases, the ulcer being situated at the junction of œsophagus and cardia in one, and at the lower end in the other two, causing partial stricture. The condition has also been described by Aurelius⁷ in 1931.

Clinical Features.—The three main symptoms are pain, hæmatemesis, and dysphagia.

I. PAIN.—This is referred usually to the lower end of the sternum, sometimes to the epigastrium. It may radiate from the point of origin downwards into the epigastrium, upwards on to the sternum, round to the back, or to the left supra-clavicular region.⁸ Barclay,⁹ in 1915, by the use of X rays after a barium meal, showed that the pain was caused by spasm of the lower end of the œsophagus, which was due to the passage of food over the ulcerated area. Œsophageal pain appears in many cases to be much worse than even the severe pain of certain gastric and duodenal ulcers; and in a case of œsophageal ulceration reported by Hurst,¹⁰ the patient became extremely neurasthenic because of it. "When a meal was brought into his room, his face became anxious and his hands shook. Several minutes elapsed before he could persuade himself to lift the fork in his trembling hand to his lips; he masticated much longer than was necessary in order to put off the moment of swallowing as long as possible, at the same time shaking his head. He had lost much weight and strength and was extremely depressed."

We should expect the pain in all cases to appear during or immediately after the swallowing of food, especially if it is simply due to spasm of the œsophagus. This is not always so, however, and, as was pointed out by Balfour,⁸ it may appear as late as two or three hours after food, so that the lesion may be confused with gastric or duodenal ulcer. He reports a case illustrating this, in which the pain and other symptoms disappeared on treating the ulcer with the application of 20 per cent silver nitrate and adhering to a bland diet, followed by dilatation of the lower end of the œsophagus. In such cases the late pain is probably due to the regurgitation of acid gastric juice during the late stages of digestion.

2. **HÆMATEMESIS.**—Only very rarely is this a rapidly fatal hæmorrhage, and when so is usually due to erosion of the aorta. More commonly there is intermittent vomiting of 'coffee-ground' material or frank red blood. Marked anæmia may develop in such cases, and in that of Zahn's¹¹ the clinical diagnosis made was one of pernicious anæmia.

3. **DYSPHAGIA.**—In the early stages of the condition this is by no means always present, but when so is due to spasm, the food being felt to stick for a few moments and then pass on. In the late stages of the condition, when fibrous stricture appears, the condition becomes much more severe, and finally complete.

Complications.—The main complications are :—

1. **PERFORATION.**—As in carcinoma of this region, perforation may occur into the pleura, pericardium, or bronchus. A case reported by Trotter,¹² in 1850, ruptured into the pericardium and caused death in twenty-four hours. Rupture into the left bronchus took place in Finlayson's case.¹³

2. **ACUTE MEDIASTITIS.**—This is an uncommon complication of the condition and is due to spread of infection from the base of the ulcer into the surrounding tissues. In Lindsay's case¹⁴ a mediastinal abscess developed and finally ruptured into the lung.

3. **DEVELOPMENT OF CARCINOMA.**—Ortmann,¹⁵ in 1901, and Tileston¹ have each described the development of carcinoma in the scars of old ulcers of the œsophagus.

Diagnosis.—Apart from the clinical signs and symptoms of the condition, which are only too often far from diagnostic, there are two methods of diagnosis at our disposal.

1. **ŒSOPHAGOSCOPY.**—By this means the ulcer may be viewed under direct vision, and it was thus that Chevalier Jackson⁴ diagnosed his active and healed-ulcer cases.

2. **X-RAY EXAMINATION AFTER A BARIUM MEAL.**—The crater of a chronic œsophageal ulcer was first visualized thus by Friedenwald, Feldman, and Zinn,⁵ in 1929. Jackson showed the presence of spasm in many of his cases, but did not succeed in showing the crater of the ulcer. Hurst,¹⁰ in 1934, demonstrated the ulcer in both of his cases by the barium meal, in one of them only after three former X-ray examinations had failed to show any lesion. In one case the cardiac sphincter was seen to be widely open, immediately above was a large shadow representing the crater of the ulcer, and, still higher, constant spasm of the œsophagus was apparent. In the second case he stresses the importance of the enormous gas bubble seen in the cardiac end of the stomach, which was due to œsophageal spasm interfering with the escape upwards of swallowed air in the same way as it interfered with the passage downwards of ingested food.

CASE REPORTS

In view of the comparative rarity of the condition, we feel that the following 8 cases of chronic simple ulceration of the œsophagus are worth recording. They have been met with in the course of 1500 post-mortems done at the Glasgow Royal Infirmary over the past four years. Cases of acute ulceration and ante-mortem digestion have, of course, been excluded; and all the cases recorded showed evidence of chronicity in the form of fibrous induration extending outwards from the ulcer and in many cases endarteritis of vessels.

Case 1.—Mrs. O'H., aged 75 years, was admitted to hospital on Jan. 14, 1936, with signs of acute obstruction of the bowels. She was operated upon as an urgent case and a cœcostomy performed. Cellulitis of the surrounding abdominal wall developed, and death took place from sepsis on Feb. 13, 1936. The patient stated in her history that she had had periodic attacks of indigestion for many years, but these had never been severe. There was no dysphagia.

POST-MORTEM REPORT.—The body was that of a woman of medium build and rather poorly nourished. A collection of pus was present between the layers of the abdominal wall in the region of the operation wound. The abdominal cavity showed only some adhesions binding the terminal ileum to the brim of the pelvis. On the anterior and posterior walls of the stomach, close to the middle of the lesser curvature, were chronic ulcers. That on the anterior wall had muscle in its base and showed marked fibrous induration around. That on the posterior wall was slightly less advanced. Each measured 1.5 cm. in diameter.

The œsophagus was opened from behind. It was normal in calibre, but at its lower end was an area of chronic ulceration extending completely round the tube. It measured 2.5 cm. in length and its lower edge coincided with the cardiac sphincter except at its left side, where it extended as a longitudinal ulcerated area measuring 2 by 1 cm. into the cardiac end of the stomach. The upper edge of the œsophageal ulcer was very poorly defined and faded off irregularly. The lower edge was more sharply seen. In the base of the ulcer the circular muscle of the œsophagus was apparent as transverse markings. Some thickening of the tissues outside the œsophagus was apparent. Besides the chronic ulcer there was also present a late ante-mortem digestion of the œsophagus showing some evidence of attempts at healing. The whole of the lower two-thirds of the tube was completely denuded of epithelium, there being simply the submucosa remaining stained light-brown with changed blood. Between this and the upper normal third of the tube was an area containing small rounded islets of epithelium which were obviously growing in an attempt to cover the denuded area.

Histological examination of the ulcerated area showed the appearances of chronic ulceration with some irregular fibrosis extending into the muscular coat of the œsophagus. The upper part of the œsophagus showed ante-mortem digestion, and at parts a thin growing edge of epithelium was seen showing attempts at healing.

The post-mortem diagnosis was cellulitis of the abdominal wall following cœcostomy; chronic ulceration of the œsophagus; ante-mortem digestion of the œsophagus.

Case 2.—Mrs. C., aged 51 years, was admitted to hospital on May 25, 1935, complaining of distension of the abdomen and marked breathlessness. Examination showed a very marked ascites, and multiple tumour masses were felt in the abdomen. Paracentesis was frequently performed, but the patient slowly went downhill and death took place on Nov. 17, 1935. There was no history of indigestion, and while in hospital the patient was fed on light diet with no particular symptoms to suggest an œsophageal lesion.

POST-MORTEM REPORT.—The body was that of a rather spare woman. The left pleural cavity contained 500 c.c. of slightly blood-stained fluid; the right contained 600 c.c. of thin purulent material with a few flakes of fibrin. Both lungs were normal. The peritoneal cavity contained 9 litres of blood-stained fluid. The peritoneal surfaces were studded with nodules of tumour tissue which had spread from a carcinoma of the uterus. The stomach and intestines were normal.

The œsophagus was opened from behind. At its lower end was an ulcer extending almost completely round it. It started a fraction of a centimetre above the cardiac sphincter and extended upwards for a length of 5 cm. On the right posterior aspect, about the middle of

the ulcerated patch, was a roughly circular area of much deeper ulceration, which extended completely through the muscular coats. At its upper edge the ulcer ceased in an irregular circinate manner, so that close observation was necessary to make out the line of demarcation. At its lower end it ceased in a very distinct, almost straight, line. Some induration of the tissues around was felt. On separating the ulcer from the adjacent mediastinal tissues, the base gave way at its deep excavated part. No direct communication was found between the ulcer and the right pleural cavity, but the septic condition of the tissues in the region of the deepest part of the ulcer suggested that the inflammatory process had spread from here through the mediastinal tissues to the right pleural cavity.

Microscopical examination of the ulcer showed involvement of the circular muscle only at most parts. Some increase of fibrous tissue was seen in the base, which contained congested vessels at places. At the deeper area of ulceration the process extended through both muscle coats and into the peri-œsophageal tissues. Much polymorphonuclear infiltration was present in the neighbourhood of this area, which gave the impression of being a more acute ulcerative process superimposed on a chronic ulcer. There was no evidence of malignant disease in the neighbourhood of the ulcer.

The post-mortem diagnosis was carcinomatosis of the peritoneum; chronic ulceration of the œsophagus; acute pleurisy.

Case 3.—Mrs. B., aged 55 years, was admitted to hospital on Feb. 5, 1933, with a hæmatemesis. For four years the patient had been troubled with periodic attacks of indigestion. Each attack lasted for a period of from a few days to three or four weeks, and were separated by relatively free periods. The pain appeared immediately after the taking of food and was referred through to the back, being felt between the shoulder blades. Flatulence was troublesome during the attacks. No vomiting had occurred until a week before her admission to hospital, when her appetite disappeared and she commenced to vomit greenish-mucoid material. High epigastric pain was present at this time and became much worse when she attempted to take food. On the morning of the day of admission she felt very faint and drank 2 drachms of brandy. She immediately felt very sick and vomited some dark blood-stained material. A short time later she vomited some fresh blood and was admitted to hospital. After admission she vomited about 5 oz. of clotted blood mixed with mucus. Examination showed nothing of note apart from slight tenderness in the epigastrium. In 1919 the patient had had oophorectomy performed, and in 1929 had had an abscess in the lower abdomen drained. Her teeth had all been removed some time previously.

Three days after admission the patient developed a pneumonic consolidation of the right lung. Blood-culture showed the presence of Type II pneumococcus. Death took place on Feb. 18, 1933.

POST-MORTEM REPORT.—The body was that of an elderly, well-nourished woman. An old midline scar was present in the lower abdomen. The cardiac muscle showed cloudy swelling. On the posterior cusp of the mitral valve were some recent vegetations of the ulcerative type. The upper lobe of the right lung was in the condition of grey hepatization. The stomach showed a certain degree of hour-glass contraction which was due to a transverse scar 5 cm. in length lying about the middle of the lesser curvature. No active ulceration was present.

The œsophagus was opened from behind. At its lower end was a chronic ulcer which extended completely round the tube and measured 5 cm. in length. Its lower edge was fairly well defined and corresponded to the cardiac sphincter. The upper edge was irregular and faded off into intact mucosa. Circular muscle showing areas of cicatrization was seen in the base. No bleeding vessel was found. The œsophagus above was of normal size.

Histologically the ulcer showed chronicity in the form of fibrous induration, extending for some distance outwards. Some endarteritis of vessels was apparent. Section of the gastric scar showed a line of well-formed old fibrous tissue.

The post-mortem diagnosis was chronic ulceration of the œsophagus; hæmatemesis; lobar pneumonia.

Case 4.—Andrew J., aged 71 years, was admitted on April 16, 1936, complaining of pain in the abdomen of two months' duration. The pain was situated in the epigastric region and was of an aching character. It tended to appear especially in the evening and was relieved by alkaline powder. The pain had no definite relationship to food, but the patient's appetite had been very much poorer than normal since its onset. There was no vomiting at any time during the illness up to the time of admission. The bowels were somewhat irregular, and on

one occasion a week or two before, the patient noted that the stool had a very black colour. Since the beginning of his illness the patient had lost over 2 st. in weight. His past health had always been very satisfactory and he did not remember having ever been previously confined to bed. Upon admission the patient showed evidence of having recently lost a good deal of weight. His jaws were edentulous and his tongue dry and furred. No epigastric tenderness was present. A blood examination showed the presence of a marked secondary anæmia, the cell-count being red blood-corpuscles 2,220,000, white blood-corpuscles 5,400, the Hb 32 per cent, and the colour index 0.72. After admission the patient periodically vomited small quantities of brown blood-stained material. On April 29, 1936, he vomited 500 c.c. of altered blood and soon afterwards collapsed and died.

POST-MORTEM REPORT.—The body was that of a male subject with a fairly large frame but showing much emaciation. The skin was very pale. The heart showed slight hypertrophy of the left ventricle. Both lungs showed marked œdema posteriorly. The prostate showed marked enlargement, due to adenomatous hyperplasia, and the urinary bladder showed hypertrophy and dilatation. The stomach contained 300 c.c. of dark-red blood-clot. No

ulceration was present in the stomach or duodenum and the blood had obviously come from the œsophageal ulcer.

In the lower 8 cm. of the œsophagus was an area of ulceration extending completely round the organ. At its upper end the ulcer extended upwards as two rounded areas with a tongue-shaped process of mucous membrane running downwards for a short distance between them. On its right side the ulceration was fairly superficial, and circular muscle could be seen in its base at places. On this side the ulcer faded off above into intact mucosa, the exact line of demarcation being very indefinite. On its left side the ulcer was much deeper over an area measuring 5 cm. in length and 2.5 cm. in breadth. The edges of this deeper area of ulceration were very steep in their inferior parts, but showed some terracing in the upper part. Near the lower edge of this part was an oval, very deeply ulcerated patch measuring 0.8 by 0.3 cm. which had as its base the intima of the aorta; and on holding the specimen up to the light a series of small cracks could be seen through which oozing of blood had taken place (Fig. 369).

Histological examination showed the appearances of chronic ulceration with much fibrous induration.

The post-mortem diagnosis was chronic ulcer of the œsophagus eroding the aorta.

Case 5.—Bernard McA., aged 53 years, was admitted to hospital on Dec. 29, 1932, suffering from hæmatemesis of four days' duration. For thirty years the patient had suffered from indigestion. The attacks lasted for weeks or months at a time and were separated by free intervals, which had lately been becoming shorter. Epigastric pain appeared from an hour to an hour and a half after the ingestion of food and lasted for a variable period, being frequently relieved by the taking of more food. Vomiting at first had been infrequent, but had been becoming more frequent during the preceding four years. Upon admission the patient was found to be an emaciated man who looked older than his years. He continued for some days to vomit 'coffee-ground' material and to pass changed blood per rectum. As he was going downhill rapidly it was decided to cauterize the bleeding ulcer. On Jan. 11, 1933, a large blood transfusion was given and, thereafter, the abdomen opened under local anæsthesia. "Kissing" ulcers were found on the anterior and posterior walls of the first part of the duodenum. That on the posterior wall was bleeding and was cauterized. A posterior gastro-enterostomy was then performed. Death took place a few hours after operation.



FIG. 369.—Case 4. Diffuse chronic ulceration of the œsophagus, which is very deep in one area and has eroded the aorta.

POST-MORTEM REPORT.—The body was that of an emaciated, elderly looking man. On the anterior and posterior aspects of the first part of the duodenum were chronic ulcers measuring 1.5 cm. in diameter. The floor of the posterior one was formed by pancreatic tissue. Slight pyloric stenosis was present and the stomach was a little hypertrophied and dilated. At its lower end the œsophagus was adherent to the tissues outside. It was opened from behind. In its lower end towards the right side was an area of chronic ulceration. This was roughly circular, measuring 3.5 by 4 cm., with edges which were rather serpiginous except the lower one, which was clean cut and ran in an almost straight line transversely across the lower end of the œsophagus. At its right side this edge lay just above the cardiac sphincter, but towards its left sloped slightly upwards from it. Growing from this edge was a small sprouting mass of what looked like granulation material. Compared with this lower edge the upper and lateral ones were indistinct and faded off into intact mucosa. The ulceration was comparatively superficial in its upper part and circular muscle was exposed in its base. In the central and lower parts, however, the process had penetrated much deeper, cicatricial



FIG. 370.—Case 5. Chronic ulcer of the œsophagus. Note the sharply defined lower edge where the ulcer is deepest. Circular muscle is exposed elsewhere.



FIG. 371.—Case 6. Chronic ulcer of œsophagus. Circular muscle and overgrown fibrous tissue are seen in the base.

fibrous tissue lying in the base. The œsophagus above the ulcer appeared slightly dilated and hypertrophied, but showed no other abnormality. (Fig. 370.)

Histological examination of the ulcer showed the appearances of chronic ulceration, there being much fibrous tissue formation with small collections of round cells. Endarteritis of vessels was seen near the surface.

The post-mortem diagnosis was chronic duodenal ulcers; hæmatemesis; chronic ulcer of the œsophagus.

Case 6.—John K., aged 69 years, was admitted on Feb. 7, 1935, complaining of pain in the upper abdomen and vomiting of six weeks' duration. For many years the patient had been troubled with indigestion, which was marked by pain coming on at a variable period after food, flatulence, and water-brash. Six weeks before admission his condition became very much more urgent. The pain and especially the vomiting became very severe, the former having no definite relationship to food, and becoming, if anything, worse after it was taken. The patient had had pneumonia in 1916 and acute cholecystitis in 1933. He was a moderate smoker and used to drink a large quantity of alcohol. This had, however, ceased some months before admission. The abdomen was slightly scaphoid and the skin dry and loose, suggesting fairly recent loss of weight. The tongue was moist with a slight white fur. All the teeth

had previously been extracted except two which appeared healthy. Slight tenderness was present over the whole abdomen. There was some anæmia present of the secondary type, the red blood-cells being 3,030,000 and the white cells 5800 per c.c. The Hb was 60 per cent. A gastric analysis showed complete absence of hydrochloric acid, with a total acidity slightly above normal. X-ray examination after a barium meal showed a moderate amount of dilatation of the stomach, but no evidence of ulceration. During the patient's residence in hospital melæna was almost constantly present. He improved slightly under treatment, but on Feb. 28, 1935, he developed signs of internal hæmorrhage from which he died on the same day.

POST-MORTEM REPORT.—The body was that of a poorly nourished looking man. The stomach was very greatly dilated, with hypertrophied wall. Immediately beyond the pylorus was a deep chronic ulcer on the posterior wall of the duodenum 2 cm. in diameter and with over-hanging steep edges. The base was formed by pancreatic tissue, and the ends of several small arteries were visible. Much fibrosis extended from the ulcer into the pancreas and lesser omentum. Black altered blood was present in the ileum and a small quantity was also found in the colon. The sigmoid loop showed long-standing diverticulitis. The gall-bladder contained twelve faceted gall-stones of the mixed type.

The œsophagus was opened from behind. Its mucous membrane was very pale in appearance. There was an area of ulceration involving the lower 5 cm. of the organ and running completely round it. The lower edge of the area lay just above the cardiac sphincter and was fairly well defined. The upper edge faded off in a series of ill-defined arcades. In this superficial ulceration were three longitudinal areas of much deeper excavation, each measuring about 1.5 cm. in breadth and showing cross striation in its base due to exposure of circular muscle fibres. At their lower edges, i.e., near the cardiac sphincter, these areas were specially deep but became much shallower above. It was the upper edges of these areas which caused the arcade-like appearance already mentioned. The wall of the œsophagus in the region of the ulceration was much thickened, fibrosed, and adherent to the structures around, so that separation was effected only with difficulty. Above the ulcer the œsophagus showed marked hypertrophy and dilatation. (*Fig. 371.*)

Histological examination of the ulcer showed it to have the characters of a chronic ulcer involving the circular muscle coat at most places, which showed varying degrees of fibrosis with collections of round-cells here and there.

The post-mortem diagnosis was chronic ulcer of duodenum with hæmorrhage; chronic œsophageal ulcer.

Case 7.—John M., aged 71 years, was first admitted to hospital on Aug. 18, 1926, complaining of loss of appetite, sickness, and vomiting of six weeks' duration. Except for occasional slight attacks of indigestion the patient had had good health previous to this time. For the first few weeks of his illness the vomiting had occurred about one and a half hours after food, but had gradually been getting much worse, and just before admission was occurring ten minutes after each meal. It was present even when he was on light diet. Abdominal pain was absent except for a slight feeling of discomfort in the upper abdomen. He had lost 1½ st. in weight during the six weeks of his illness. The patient admitted that he had previously taken a fairly large quantity of alcohol every week. He smoked 3 oz. of tobacco per week. Upon admission the patient was found to be a rather thin elderly man. There was evidence of recent loss of weight. A neoplasm of the stomach was suspected, but X-ray examination of the gastro-intestinal tract was negative. He was put on light diet, his sickness ceased, and he commenced to put on weight. He was discharged on Sept. 16, 1926, feeling very well and with no return of symptoms.

On Sept. 1, 1931, the man was readmitted to hospital. He stated that shortly after his discharge five years before his stomach had recommenced to trouble him. This took the form of periodic attacks of epigastric pain and vomiting, the pain appearing at a variable period after food, often coming on soon after its ingestion. He found that heavy foods such as potatoes and meat aggravated his symptoms and he had been avoiding these. For the five days preceding admission the symptoms had become very urgent, the pain in the epigastrium being very severe, and hiccup was almost constantly present. The day before admission he vomited a quantity of 'coffee-ground' material. Examination showed the patient to be a rather spare, elderly man. Constant hiccup was present. The patient had no teeth. The breath was foul and the tongue thickly coated with a dirty, brownish fur. Deep pressure in the epigastrium elicited some tenderness but no mass was palpable. The lungs and urinary

and nervous systems were normal. The patient appeared a little confused and gave only a very indefinite account of the symptoms. He improved under very light diet, and on Sept. 8 the sickness had ceased and the hiccup was only occasionally present. He was being given 3 pints of milk and a $\frac{1}{2}$ pint of Benger's food daily. On admission the stools contained obvious melæna, but on Sept. 12 it was only found on careful chemical examination. Examination of vomited material on Sept. 13 showed free hydrochloric acid (titrated with N/10 NaOH) 50; total acidity, 78. On Oct. 19, 1931, the improvement had persisted and the patient's mental condition was much clearer. He complained that his food stuck near the lower end

of his sternum after he had swallowed it. An X-ray examination after a barium meal was carried out and the report was "there is obstruction of the œsophagus, the appearance being highly suggestive of neoplasm". An œsophagoscopic examination was carried out on Dec. 2, 1931. No carcinoma of the œsophagus or fundus of stomach was seen. The walls of the œsophagus appeared rather closer together than normal, suggesting an extrinsic pressure on it, but no decisive finding was obtained. The patient was now put on 5 min. of tincture of belladonna three times daily.



FIG. 372—Case 7. Diffuse ulceration of œsophagus, the wall being greatly thickened by fibrosis. The lower edge is fairly well defined, but the ulceration fades off in the upper part. Probes are in two vessels which had been bleeding.



FIG. 373—Case 8. Peptic ulcer of œsophagus. Note the deep overhanging edge and the tongue-shaped process of heterotopic gastric mucosa extending upwards between the lateral edges of the ulcer. The base is formed by the pleural surface of the left lung.

This improved his œsophageal condition and he was able to swallow his food normally after this. On Dec. 7, 1931, he was discharged from hospital feeling very well.

On Aug. 30, 1932, the patient was admitted to hospital for the third time. He complained of hæmatemesis and epigastric pain of one day's duration. He said that he had been keeping fairly well since his discharge from hospital less than a year previously. Four days before his present admission he developed epigastric pain accompanied by marked nausea. Alkaline treatment only helped him slightly. On the day preceding admission the pain was very much more severe and the patient vomited brownish material which contained blood. Examination showed him to be a moderately well nourished elderly man. His complexion was florid and his mucous membrane of good colour. He complained of vague high abdominal pain but no rigidity was present. Soon after his admission he had a copious motion which contained

altered blood. While in hospital he had various attacks of vomiting, the vomitus consisting of partly digested food. On Sept. 11 he had two attacks of hæmatemesis which weakened him considerably. He also complained of pain high up in the epigastrium shooting through to the back. On Sept. 27 it was noted that he continued to vomit about once daily and that the vomited material always contained some altered blood. This vomiting appeared to be unaffected by any medicinal treatment. His general condition became poorer, he became confused mentally, and died on Nov. 5, 1932.

POST-MORTEM REPORT.—The body was that of an adult man of rather poor nutrition. The heart and lungs showed no abnormality apart from senile degenerative changes. The stomach showed slight dilatation, but no ulceration, recent or old, was present. No other abnormality was found in the abdomen.

Externally the œsophagus felt greatly thickened in its lower half and was firmly adherent to the tissues outside, so that it could not be completely separated from them. It was opened from behind. The lower two-thirds of the organ showed very marked thickening of its wall due to replacement by firm fibrous tissue, and measured 7 mm. in thickness at parts. The mucous membrane was completely absent in the lower half. The ulcerated area ceased abruptly in a straight line at the cardiac sphincter, but at its upper part it faded gradually off into intact mucosa, there being a 'transitional' area where islets of epithelium started to appear, showing some attempt at healing. The base of the ulcer consisted mainly of cicatricial fibrous tissue, but at parts the cross striation of the circular muscle coat was apparent. Two small perforations into vessels were found in the middle of the ulcerated patch. Blood exuded from the lower one on slight pressure. Just outside the œsophagus, on the left side 3 cm. above the cardiac sphincter, was an enlarged gland the size of a bean containing much anthracotic pigment. (*Fig. 372.*)

Microscopical examination of a piece of tissue taken from the middle of the ulcer was carried out. The remains of longitudinal muscle were present at one end of the section. It showed a well-marked increase of fibrous tissue between its fibres. The fibrous tissue was dense and hyaline at parts but became more cellular and of more recent origin nearer the surface. Many of the vessels showed marked endarteritis obliterans. There was no evidence of malignant or syphilitic change. In the fibrous tissue deep to the muscular coats numerous lymphoid follicles with prominent germ centres were seen.

The post-mortem diagnosis was chronic ulcer of the œsophagus with hæmorrhage.

Case 8.—Patrick C., aged 58 years, was admitted on June 8, 1935. For years his stomach had troubled him, there being an indefinite history of pain after food accompanied by occasional vomiting. An X-ray photograph was taken, the suspected diagnosis being peptic ulcer; but no abnormality of the stomach or duodenum was seen. For some months before admission the patient's disability had become extreme. He was afraid to take food because of the pain, had become very neurasthenic, and had taken to alcohol as a means of relieving the pain. There was the occasional vomiting of 'coffee-ground' material. Anæmia had become very marked, and he was admitted to hospital with a condition of spasm of the vessels of the fingers resembling Raynaud's disease. Upon admission the patient was very weak and breathless. He admitted having drunk half a bottle of whisky an hour or so before admission. He died fairly suddenly four hours later.

POST-MORTEM REPORT.—The body was that of a middle-aged male in the last stages of emaciation. In the posterior aspect of the lowest lobe of the right lung was an area of red hepatization. A less advanced degree of pneumonia was also present in the middle lobe. All the organs of the body showed evidence of inanition. The marrow of the femur showed a well-marked erythroblastic reaction.

Marked thickening was present in the tissues around the lower end of the œsophagus, so that carcinoma was suspected on external examination. The organ was opened from behind. At its lower end, 1 cm. above the cardiac sphincter, was a deep oval ulcer, measuring 5.5 by 4 cm., almost completely encircling the circumference of the organ and leaving only a small patch of intact mucosa 1.5 to 2 cm. in breadth. The edges of the ulcer were very steep and overhanging at parts. The base was firm and infiltrated, and at its left side was composed of the pleural surface of the left lung, which showed some fibrosis at this part. Gentle manipulation broke down the adhesions binding the ulcer to the pleura, causing a direct perforation into the left pleural cavity, showing how the patient had been living on the verge of a precipice for some time. Closer examination of the mucous membrane in the region of the ulcer

showed the presence of a remarkable state of affairs. The intact mucosa separating the lateral edges of the ulcer was found to be heterotopic gastric mucosa which extended as a tongue-shaped process of well-preserved tissue upwards from that of the fundus of the stomach. (Fig. 373.) The two important features were the close resemblance of the ulcer to the typical chronic ulcer of the pyloric region of stomach or first part of duodenum and the presence of the heterotopic mucosa.

Microscopical examination showed marked fibrosis spreading from the base of the ulcer, with complete destruction of the muscle coats in the neighbourhood. On the surface of the ulcer was a thin layer of necrotic material infiltrated with polymorphs and fibrin. Deep to this was a fairly cellular granulation tissue with many fibroblasts. Beneath, this fibrous tissue became more dense and avascular. The heterotopic gastric mucosa bore a resemblance to that found normally towards the pyloric end of the stomach, the glands being fairly short and wide. Oxyntic cells were present, but were comparatively few in number.

The post-mortem diagnosis was chronic peptic ulcer of the œsophagus; advanced inanition; lobar pneumonia.

Table I shows the main features of the 8 cases described in detail above.

Table I.—SHOWING MAIN FEATURES OF 8 CASES OF CHRONIC PEPTIC ULCER OF THE ŒSOPHAGUS

SEX	AGE	SYMPTOMS	ŒSOPHAGUS	GASTRIC OR DUODENAL ULCERATION	CAUSE OF DEATH
Female	75	Periodic attacks of indigestion	Chronic ulcer with ante-mortem digestion	Chronic gastric ulcers	Cellulitis of abdominal wall after cæcostomy
Female	51	No history of indigestion	Chronic ulcer more acute at one part	None	Carcinomatosis of peritoneum, with terminal mediastinitis and empyema due to œsophageal ulceration
Female	55	Indigestion for 4 years: hæmatemesis on admission	Chronic ulcer	Scar of old ulcer across lesser curvature	Lobar pneumonia following hæmatemesis from œsophageal ulcer
Male	71	Indigestion for 2 months	Chronic ulcer of rounded type	None	Hæmorrhage from eroded aorta
Male	53	Indigestion for 30 years	Chronic ulcer of rounded type	Chronic ulcers of duodenum	Death following operation on duodenal ulcers
Male	69	Indigestion for many years	Chronic ulcer	Chronic ulcer of duodenum	Death due to hæmorrhage from duodenal ulcer
Male	76	At first like gastric condition. Later dysphagia	Very chronic ulceration with marked fibrosis	None	Death from constant bleeding from ulcer
Male	58	Indigestion for many years, much pain latterly. Very neurasthenic and alcoholic because of pain	Typical peptic ulcer of œsophagus with heterotopic gastric mucosa	None	Pneumonia following advanced inanition from hæmorrhage and starvation due to ulcer

DISCUSSION

Certain important features emerge in a study of the eight cases which we have just described.

a. Age Incidence.—All the patients were over 50 years of age, the average age being 63·5 years. It is to be noted, however, that the ulcers had been unsuspected during life and some had probably been present for years, so that the true age incidence will be actually lower than this post-mortem figure.

b. Sex.—Five of our patients were males and three were females, so that the sex incidence appears to be slightly higher on the male side.

c. Coincident Ulceration.—In four of the cases ulceration was also present in the stomach or duodenum, the patient having apparently the so-called 'ulcer diathesis'.

d. Symptoms.—In two of the cases the patients had no notable dyspeptic symptoms. In the others, indigestion had been present for a variable period of time, usually for years. In two of these the symptoms were most likely due to the duodenal ulceration which was also present. In one the gastric ulcer had been healed for a long period of time, leaving an avascular fibrous scar, so that the late symptoms were almost certainly due to the œsophageal ulcer. In the remaining three cases the severe dyspeptic symptoms were undoubtedly due to the œsophageal ulceration. It is worth noting that in all of them the symptoms were referred to the stomach and duodenum, not only by the patient, but by the physician; and in only one of them, and that at a late stage when fibrous stricture was taking place, was the œsophagus suspected as the cause of the trouble. In four of our cases hæmatemesis was present, but in only two of them was this a marked feature.

e. Cause of Death.—In five of the cases the œsophageal ulceration could be said to be at least an important factor in causing death. In two of these the actual cause was lobar pneumonia, but in both the ulcer was undoubtedly the exciting factor, in the one case being superimposed on the very advanced inanition which it had produced in the patient, and in the other following a hæmatemesis from the ulcer. In one case the immediate cause of death was empyema and mediastinitis which had spread from the base of the ulcer. In this case chronic ulceration had been present for some time and a more acute process had become superadded, most likely as the result of the extremely debilitated condition of the patient following the malignant cachexia. In another case death was the result of recurring bleedings from the ulcer. In the last case death was due to erosion of the thoracic aorta by the ulcer.

f. Position of the Ulcer.—In all the cases the ulcer was situated at the lower end of the œsophagus, and in many of them its lower edge was sharply limited by the cardiac sphincter.

g. Appearance of the Ulcer.—There appears to be two different types of ulcer found in this region and we shall later suggest two different etiologies:—

1. In the first type the ulceration is fairly superficial in type. It may remain thus as in Cases 1, 3, 6, and 7, and in the last case the extreme amount of fibrous thickening of the wall which may ensue should be noted. On the other hand, it may become much deeper at one area, an ulcer within an ulcer as it were, and, as in Case 2, cause mediastinitis and empyema, or, as in Case 4, erode the aorta. This type of ulcer involves the area of œsophagus immediately above the cardiac sphincter.

The lower edge is in most cases clean cut and unhealing, whereas the upper one is indefinite, often circinate, and fades off into intact mucous membrane.

2. In the second type the ulcer is localized, is very deep and penetrating, and in fact has the typical appearance of a very chronic gastric or duodenal ulcer. The presence of gastric heterotopia in the region of the ulcer is to be noted, and, clinically, the extremely urgent symptoms causing profound neurasthenia and inanition.

h. Diagnosis.—Repeated and very careful œsophagoscopic and X-ray examinations appear to be necessary to demonstrate the condition, and in one of our cases narrowing of the œsophagus was the only definite lesion diagnosed by the œsophagoscope, although at the autopsy a short time later a very advanced ulcerated and fibrous condition of the tube was seen. We have already discussed the X-ray diagnosis of the lesion by visualizing the crater after a barium meal. From the study of our cases we feel that this will be the exception rather than the rule, and that the most certain diagnostic feature is likely to be spasm in the region of the ulcer, for in our series of cases very few had craters large enough to fill with barium.

ETIOLOGY

The two types of ulceration which we have described are so different in their appearances that we are tempted to look for two different causes.

1. The shallow, more diffuse type of ulceration conforms in its position, widespread character, and abrupt cessation at the cardiac sphincter to the acute form of ulceration of the œsophagus called 'ante-mortem digestion'. We have examined specially the œsophaguses from a fairly large number of cases and have concluded that, whereas the acute fulminating type of ante-mortem digestion is fairly rare, the slighter degrees are not uncommon, and may very likely be frequently recovered from, in some cases completely, in others, as we shall suggest, leaving an area of chronic ulceration.

Like burns, ante-mortem digestion can be divided into degrees of severity, examples of all of which have been seen post mortem. The first stage is one of congestion of the œsophagus with no destruction of the mucous membrane. In the second, the mucous membrane has disappeared at places, leaving, however, intact islets of epithelium between. In the third degree the mucous membrane has disappeared completely over a large area of gullet, almost always its lower third. In the fourth or last degree the whole thickness of the wall is necrotic and perforation may take place into the pleural sac. Death in such cases is, of course, invariable. In the second degree of the condition there are islets of epithelium left corresponding to the interpapillary processes of skin in a third-degree burn; and should the patient recover from his primary illness, these will act as centres from which epithelium will grow to cover completely the denuded area. Such healing cases are sometimes seen post mortem. To the naked eye the islets appear rounded off at their edges, resembling thin patches of white paint on the raw surface, which has by this time lost the dark sanguineous appearance of the acute stage of the condition (*Fig. 374*). Microscopically the epithelium is seen growing as a thin layer over the denuded patch, mitotic figures being seen in the stratum mucosum near the growing edge.

Consider the third degree of ante-mortem digestion—the stage where there is

complete destruction of the mucous membrane of the lower end of the œsophagus, corresponding to a fourth-degree burn. If such a case recovers from the primary illness what will happen to the affected part? The necrotic material will separate and the mucous membrane at the edge of the area commence to grow in to cover the denuded area, but there are no epithelial buds left to fill in the central part, and if we carry our analogy with a burn of the skin still further, we can see how difficult it would be for such an area to be completely epithelialized—how healing usually takes place so far and then ceases until skin-grafts are applied. Still more difficult will it be for healing to take place in the case of the œsophagus, whose mucous membrane is being constantly irritated by food, and on occasions also by regurgitated acid gastric juice. Thus, a stage will be reached when the acute ulcer is replaced by a chronic ulcer of the diffuse and fairly shallow type like many we



FIG 374—Healing ante-mortem digestion of the second degree, showing the rounded edges of the growing islets of epithelium.

have described. The upper edge which is farthest from the irritating gastric juice will show more healing than the lower edge which is in close proximity to the gastric contents; the lower edge will be sharp and clean cut, showing little healing as compared to the ill-defined healing upper one.

As an additional proof of this etiology, we would point out the association of four of our cases with the 'hyperchlorhydric' conditions of gastric and duodenal ulceration; and also that in one of our cases there was, along with the chronic ulceration, a recent ante-mortem digestion of the œsophagus above the chronic lesion.

2. Our second type of ulceration differs very markedly from the first and is shown in its typical form in *Case 8*. It resembles the chronic ulcer found in the pyloric end of the stomach or the first part of the duodenum, and is marked by depth rather than diffuseness. The symptoms also appear to

be more urgent and serious in such cases. A clue as to its origin was given by the finding of a large patch of heterotopic gastric mucosa beside the ulcer. Heterotopic mucosa was present in Stewart and Hartfall's case³ and in Fraenkel's¹⁶ and Tileston's¹ cases. When the patches are small and the amount of acid secreted correspondingly little, it will be rapidly diluted by saliva and cause no harm. Thus heterotopic mucosa is not infrequently found in the œsophagus with no coincident ulceration. If the patches are more extensive, and if there is some degree of spasm of the cardiac sphincter superimposed, the accumulation of this acid secretion in the lower part of the œsophagus will set up an irritative, and later an ulcerative, condition. That is the view usually held as to the association of peptic ulcer of the œsophagus with gastric heterotopia. If that be the correct explanation, we should expect the ulceration to be rather of the diffuse type which we have already described instead of the localized crateriform type. Thus, while not denying that such is the correct explanation, especially where the

heterotopic mucosa occurs in the upper part and the ulcer in the lower part, we also suggest from the appearances of our own case that the ulcer may actually start in the heterotopic mucosa itself, which, as is well known in the case of Meckel's diverticulum, shows a marked tendency to ulcerate when it finds itself in a foreign environment.

SUMMARY

1. Eight cases of chronic ulceration of the lower end of the œsophagus are described.
2. They are discussed and tabulated to show their main features.
3. Two main types are distinguished and the etiology of each of these types is discussed.

My thanks are due to Professors Harrington and Burton and Drs. Middleton, Muir Crawford, and Henderson for the use of the clinical notes of their cases, and above all to Professor J. Shaw Dunn for his very kind and willing help and advice at all times.

REFERENCES

- ¹ TILESTON, W., *Amer. Jour. Med. Sci.*, 1906, cxxxii, 240.
- ² STEWART, M. J., *Jour. Pathol. and Bacteriol.*, 1929, Jan., xxxii.
- ³ STEWART, M. J., and HURST, A. F., *Gastric and Duodenal Ulcer*, 1929, 498. London: Oxford University Press.
- ⁴ JACKSON, CHEVALIER, *Jour. Amer. Med. Assoc.*, 1929, xcii, 369.
- ⁵ FRIEDENWALD, J., FELDMAN, M., and ZINN, W. F., *Amer. Jour. Med. Sci.*, 1929, clxxvii, 1.
- ⁶ EUSTERMANN, G. B., MOERSCH, H. J., and CAMP, J. D., *Med. Clin. N. Amer.*, 1930, xiv, 565.
- ⁷ AURELIUS, J. R., *Amer. Jour. Roentgenol.*, 1931, xxvi, 696.
- ⁸ EUSTERMANN, G. B., and BALFOUR, D. C., *The Stomach and Duodenum*, 1935. Philadelphia and London: W. B. Saunders Co.
- ⁹ BARCLAY, A. E., *Proc. Roy. Soc. Med. (Electro-Therap. Sect.)*, 1915, viii, 96.
- ¹⁰ HURST, A. F., *Jour. Amer. Med. Assoc.*, 1934, cii, Feb. 24.
- ¹¹ ZAHN, F. W., *Rev. méd. de la Suisse rom.*, 1882, ii, 144.
- ¹² TROTTER, J. W., *Trans. Pathol. Soc. Lond.*, 1850-52, iii, 316.
- ¹³ FINLAYSON, J., *Glasgow Med. Jour.*, 1883, xix, 313.
- ¹⁴ LINDSAY, J. A., *Dublin Jour. Med. Sci.*, 1899, cvii, 266.
- ¹⁵ ORTMANN, K., *Münch. med. Woch.*, 1901, xlviii, 387.
- ¹⁶ FRAENKEL, A., *Wien. klin. Woch.*, 1899, xii, 1039.

RENAL RICKETS

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DURING the last quarter of a century it has become possible, largely as a result of improved X-ray technique and an extended use of biochemical analysis, to clarify many formerly obscure abnormalities in bone development, particularly perhaps that group designated somewhat loosely as late or adolescent rickets. It is probable that a certain number of these cases were merely examples of hunger osteomalacia, especially those occurring in some areas after periods of famine, the most important recent examples of which were supplied by the post-war period in Central Europe. There remains a group of cases in which rachitic changes appear during later childhood or adolescence, or which persist from infancy into this period in spite of treatment, and which, taken as a group, are not particularly rare, presenting from time to time a diagnostic problem to every alert clinician. This group necessarily includes a few cases of ordinary infantile or vitamin D deficiency rickets in which the nutritional deprivation has remained uncorrected or has made its appearance for some reason at an abnormally late period in the child's life. It also includes two forms of so-called rickets in which certain entirely new factors are concerned, namely, coeliac and renal rickets. With the latter must be considered certain forms of primary hyperparathyroidism in which secondary renal changes occur. Coeliac rickets has been admirably dealt with by Parsons,¹ and will not be further considered here beyond remarking that it is essentially a low-calcium form of rickets due to long-continued loss of vital elements in the stools.

The problem of the pathogenesis of renal rickets remains incompletely solved, and has been complicated during recent years by the discovery in certain cases of enlarged parathyroids. The issue has thus become entangled in the far-flung net of the endocrinologist, who would like to range the disease alongside that type of hyperparathyroidism to which reference has already been made. In view of the rarity of the condition, and of the uncertainty which still surrounds the question of its etiology, it is important that every case in which the diagnosis can be established should be reported as fully as possible.

CASE REPORT

HISTORY.—The patient, a boy of 14, was admitted to Southmead Hospital on April 20, 1936, exhibiting most of the typical features of renal rickets. At birth he was stated to have weighed only 4 lb. He was always regarded as delicate, and remained stunted in growth, but exhibited no deformities until well past the age of seven. He had long suffered from thirst and polyuria, first noticed between two and three years of age. He had had measles at two, and at the age of seven contracted nasal diphtheria, for which he was treated at a fever hospital for thirteen weeks.

Deformities of the legs and feet were first noticed when he was nearly 8, and had become progressively worse, particularly during the last year. The deformities prevented

him from running or playing games, but he had ridden a bicycle right up to the day of admission, and was in other ways physically active. He had never been able to talk properly, and had always been extremely deaf. Notwithstanding all this, his parents, who were very fond of him, insisted that he was as mentally alert as any of their five other children had been at a comparable age period. His diet had always been adequate and varied, except that he disliked and rarely touched meat. The stools were normal.

The mother, aged forty-six, gave an interesting obstetrical history which may be recorded as follows:—

Date of pregnancy	1910	1912	1914	1916	1918	1921	1922	1923	1923
Sex of offspring	♂	♂	♀	♀	♀	M	♂	M	M
Age of offspring	26	24	22	20	18	—	Patient aged 14	—	—

(M = miscarriage)

The child's parents both appeared to be in perfect health, and there were five other living children, none of whom exhibited the slightest deformity or under-development. The Wassermann reactions and Kahn tests on both father and mother were negative, while their serum calcium and phosphate estimations were normal.

ON EXAMINATION.—The boy weighed 4 st. 7 lb. and was 4 ft. 1 in. in height, figures which are roughly equivalent to those of a boy of eight or nine. He was of a sallow muddy complexion with freckling of the forehead and round the nose. The hair was coarse and dry and somewhat sparse. There was a moderate anæmia of the mucous membranes. The malar bones were unduly prominent, giving a depressed appearance to the bridge of the nose, which was rather squat and broad. The eyes appeared rather sunken by comparison, and were in addition somewhat heavily lidded, so that he had a perpetually sleepy appearance. Another striking feature about the face was the prominence of the superior maxillæ, so that the lower jaw was definitely receding (*Fig. 375*). (It is possible that this peculiar facial conformation represents a type of deformity occasionally occurring in renal rickets, as a somewhat similar appearance has been referred to in a case described by Ellis and Evans.²)

The whole appearance was suggestive of considerable mental defect, but his intelligence was difficult to assess owing to the deafness and a rather gross speech defect of the lalling type. He was certainly able to lip-read quite well, and his ability to express himself by a wealth of sign and gesture hinted at an underlying mental activity utterly belied by his very unprepossessing exterior.

Both testes, though small, had descended into the scrotum, but secondary sex characteristics were rather infantile. Nothing abnormal was detected in the cardiovascular system. Blood-pressure: 120/75. No definite thickening of peripheral vessels; eye grounds normal. The kidneys were not palpable.

Deformities.—The most striking abnormality was due to a flexion of both hips, with marked compensatory lordosis of the lumbar spine (*Fig. 375*). This caused a rather waddling gait reminiscent of bilateral congenital dislocation of the hips. Even with the lordosis the effect of the flexed hips was never fully overcome, so that he was not able to stand upright properly. At the ankles there was an outward bending of the lower ends of tibia and fibula on both sides, with a resulting valgoid displacement of the feet (*Fig. 376*). There was moderate genu valgum and a well-marked rickety rosary, while a swollen metaphysis could be palpated at the ends of most of the long bones.

X-ray Examination.—Radiographs of the whole skeleton were taken, and revealed characteristic changes of renal rickets. There was a generalized osteoporosis associated with florid rachitic changes at the extremities, the wide-cupped metaphyses exhibiting that peculiar fluffy or woolly appearance which is so constantly seen in severe cases, and which, as Brailsford³ has rather aptly suggested, looks as if the cancellous bone has undergone a mucinous degeneration. Many of the bones were bent and contorted, particularly the upper end of the femur and the lower ends of the tibia and fibula. The necks of both femurs showed what appeared to be an almost complete separation from the upper epiphyses (*Fig. 377*), while at the lower ends of the tibia and fibula on both sides the outward rotation of the bones had caused a partial sheering off at the softened epiphysal junction, producing the

extreme valgoid deformity to which reference has already been made (*Fig. 378*). Gross changes of the woolly type were present at the lower end of the radius and ulna, with subperiosteal erosion extending along the shafts (*Fig. 379*). The skull showed a remarkable degree of thickening and rarefaction with considerable stippling and 'honeycombing' (*Fig. 380*).



FIG. 375.—Lateral view of patient, showing facies and extreme lordosis.



FIG. 376.—Front view of patient, showing facies and valgoid deformity of feet.

RENAL INVESTIGATIONS.—The urine was pale and copious, of uniformly low specific gravity (1000–1004), and contained a trace of albumin and an occasional hyalogramular cast. Urea-concentration tests showed literally no concentrating power, viz., before urea, 0.85 per cent; one hour after, 0.85 per cent; two hours after, 0.8 per cent; three hours after, 0.85 per cent. Water excretion test: after 1 pint, 13 oz. excreted in 4 hours. Maximum specific gravity 1004, minimum 1000.



FIG 377.—Radiograph of hip-joints showing rachitic changes and false appearance of epiphysial displacement.



FIG 378 —Radiograph of lower end of right tibia and fibula showing collapse of metaphysis and rotation and partial separation of epiphys.s.



FIG. 379 —Radiograph of left wrist showing gross rachitic change of the woolly type, with subperiosteal eros on.

BLOOD CHEMISTRY.—Blood-urea 318 mg. on April 29, 1936. On June 15, 1936, a day prior to death, the blood-urea had risen to 397 mg. Serum calcium 13.6 mg. per cent; one month later, 12.5 per cent. Serum phosphates 6.5 per cent; one month later, 5 mg. per cent. Phosphatases 59.6 units (April 29, 1936); 51.1 units (June 15, 1936). Wassermann and Kahn reactions gave a negative result.

COURSE OF THE DISEASE.—In spite of the startling degree of renal inadequacy as evidenced by the blood-urea and renal function tests the boy to all intents and purposes appeared to be in comparatively good health, was bright and cheerful, and frequently amused his fellow patients by a versatile display of pantomime with his hands. The uræmic finale which supervened came with quite unexpected suddenness, beginning with drowsiness and twitching, passing on to convulsions and coma, and terminating fatally in twenty-four hours.

POST-MORTEM EXAMINATION.—Permission for a limited post-mortem was obtained only with great difficulty, so that our examinations were confined to one femur, one kidney, and the parathyroids.



FIG. 380.—Radiographic appearance of skull showing thickening, rarefaction, and 'honey-combing'.

The Femur.—There was a striking disparity between the apparent radiographic appearance (*Fig. 377*) and the finding at post-mortem (*Figs. 381, 382*), for whereas the radiograph suggested a slipped epiphysis at the hip-joint the actual lesion was a collapse of the metaphysis of the neck. In this situation the ordinary cortical and cancellous bone is replaced by a mixture of fibrous tissue and cartilage, in which some bony spicules can be made out. The lines of ossification at both ends of the bone are greatly thickened and irregular, and the hyaline cartilage of the epiphysal line of the head is continuous with the mass of cartilage and fibrous tissue forming the collapsed neck of the bone. The epiphysal line of the great trochanter is also greatly increased in depth, extending deeply into the metaphysis and also joining up with the large mass of fibro-cartilage already mentioned. The lower end of the femur presents no gross deformity, though the epiphysal line is thickened and the bony cortex here thinned and deficient. The thinning of the bony cortex is seen most pronouncedly in the head, where the somewhat thickened articular cartilage appears to have no underlying layer of bone but to be attached directly to the trabeculae and spicules of the cancellous portion. In the shaft the cortex is spongy and soft, but the marrow cavity appears normal in extent and not unduly vascular.

Microscopically the femoral head shows thinning of the cortical layer of bone (*Fig. 383*). In places calcification and osseous structure is present only as a thin encrustation on the deep aspect of the cartilage, while over large areas it is completely absent. The microphotograph illustrates well the evidences of simultaneous bone formation and absorption as represented

by areas where osteoblasts in rows are apparently concerned with formation of freshly calcified cartilage, while in close proximity to these are bony laminæ showing the occasional lacunæ in which large osteoclasts are producing the typical clear-cut absorption indentations. The

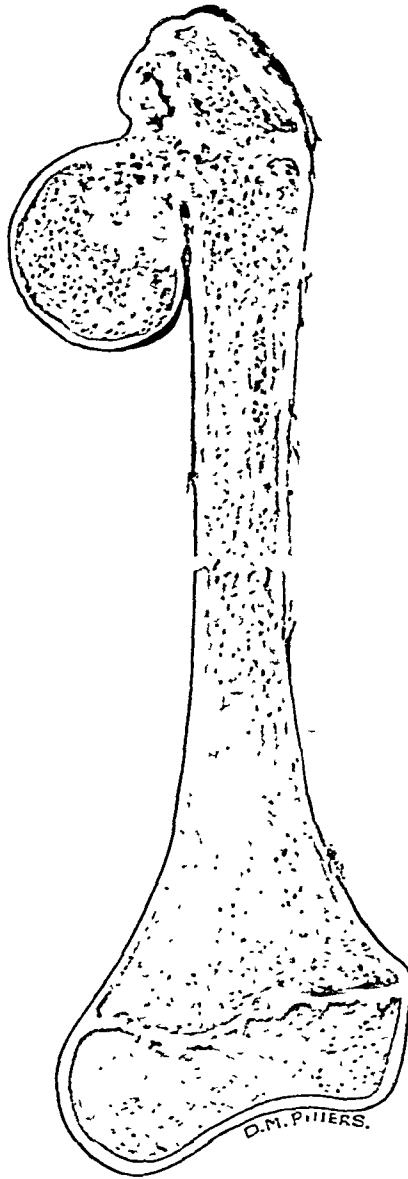


FIG. 381.—Femur, showing the metaphysial collapse of the neck and widened lines of ossification.

trabecular bony spicules show similar evidences of bone formation and absorption, without any signs of osteoid tissue, and the inter-trabecular spaces are occupied by a loose,

well-vascularized, fibro-fatty tissue which becomes more cellular and firm at the periphery. There is no evidence of any hæmopoiesis.

Sections of the epiphysial cartilage of the head (*Fig. 384*) show, on the metaphysial side

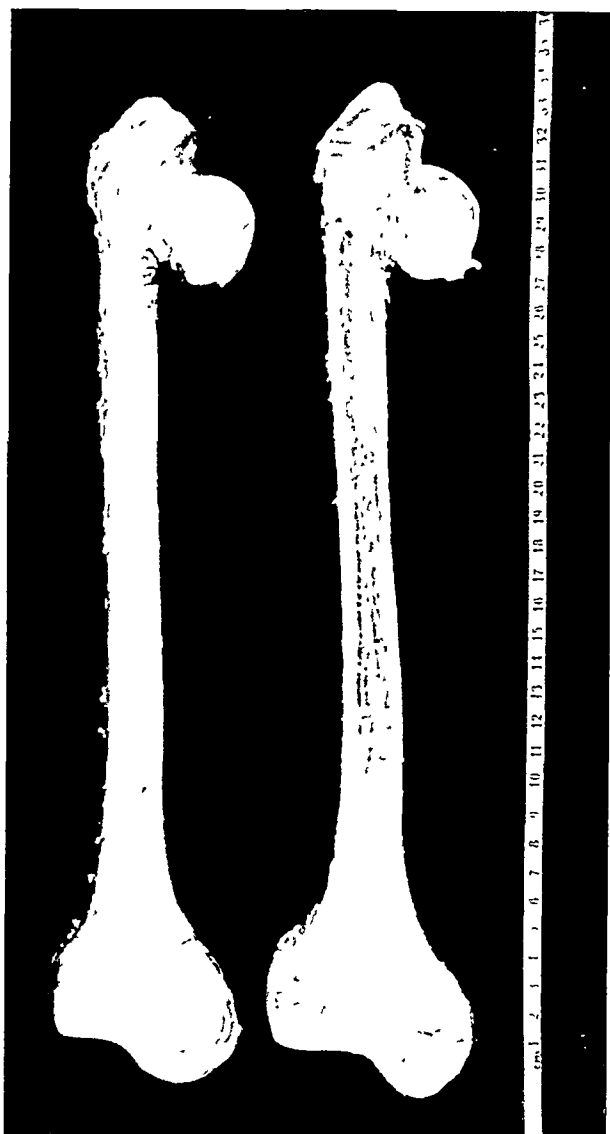


FIG. 382.—Whole femur after longitudinal section.

of the greatly thickened band of hyaline cartilage, an absence of the normal columns of hyperplastic cartilage cells merging into osteoid and bony spicules. Here, as in the cortex of the bone, evidences of absorption predominate. The bony spicules are firm and well formed, and there is practically no osteoid structure. The inter-trabecular matrix is more fibrous than normal.



FIG. 383.—Microphotograph of cortex of head of femur showing the articular cartilage and the scantiness of the cortical layer of bone. ($\times 50$.)



FIG. 384.—Microphotograph of the diaphysal aspect of the epiphyseal line of the head of the femur, showing the complete lack of the usual proliferating columns of cartilage cells. ($\times 50$.)



FIG. 385.—Microphotograph of fibrotic tissue constituting the collapsed metaphysis of the neck of the femur. ($\times 50$.)

The microscopical structure of the softened and collapsed metaphysis of the neck is seen in *Fig. 385*. Bony spicules are few, and merge directly, without the intervention of any layers of osteoid, into a fairly dense fibrous tissue in which are numerous large vascular channels. Such activity as is evinced in connection with the bony trabeculae is almost entirely that of absorption by osteoclasts.

To summarize: The changes in the bone are essentially those of cessation of growth with pronounced bony absorption and demineralization, which have led to weakening at all points of strain, and hence to the broadened splayed epiphyses, and more particularly to the extensive metaphysal collapse, with attempts at compensation by overgrowth of fibro-cartilage from the epiphysal line and articular surfaces. The changes resemble rickets only superficially, and present none of the characteristic microscopical structures associated with avitaminosis D.

The Parathyroids.—As seen in the coloured plate (*Fig. 386*) all four parathyroid glands were enlarged to many times their normal size, their actual measurements being $10 \times 6 \times 4$ mm. and $15 \times 6 \times 4$ mm. for the upper pair, and $15 \times 8 \times 6$ mm. and $11 \times 10 \times 5$ mm.



FIG. 386.—Posterior view of the thyroid gland to show the position and size of the four hyperplastic parathyroids.

for the lower (the average normal size is about $5 \times 3 \times 2$ mm.). In each case the glands presented a homogeneous appearance, and they were regarded as hyperplasias and not as adenomas.

Their microscopical structure presented no abnormal features. The cells were mainly of the principal type arranged in the usual columns and cords, with, here and there, definite alveolar arrangement with appropriate elongation of the cells. The oxyphil cells were not numerous, but also not unusually sparse considering the age of the patient. These oxyphil cells usually occurred in small clumps of two to five, but in a few places formed much larger congeries. One unusual feature presented itself in the section of one of the lower glands, in that the pericapsular tissue consisted of thymus gland. This probably represents an ectopic position of the thymus, though in cases in which the thymus is enlarged it is not unusual to find it extending to the base of the thyroid gland. By reason of the limited nature of the post-mortem examination in this case the size of the thymus was not observed, and the finding of thymic tissue in this situation was quite unexpected. The Hassall corpuscles in this fragment of the gland were very large, and most of them were partially or completely calcified.

The Kidneys.—The one kidney which was obtained for examination was greatly reduced in size and pale in colour (*Fig. 387*). The capsule was slightly adherent, but the surface was

diffusely and evenly granular (*Fig. 388*), the pitting and excrescences being coarser than is seen in chronic nephritis, and resembling closely the irregularity of advanced renal changes of essential hypertension. The cut surface also was pale. The cortex was narrow, the

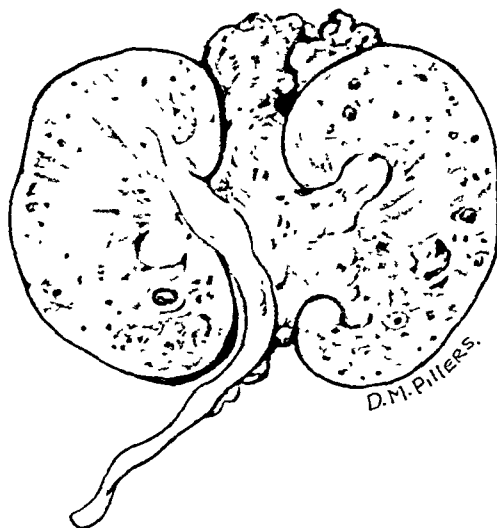


FIG. 387.—Kidney: cut surfaces showing the generalized fibrosis and loss of demarcation between cortex and medulla.

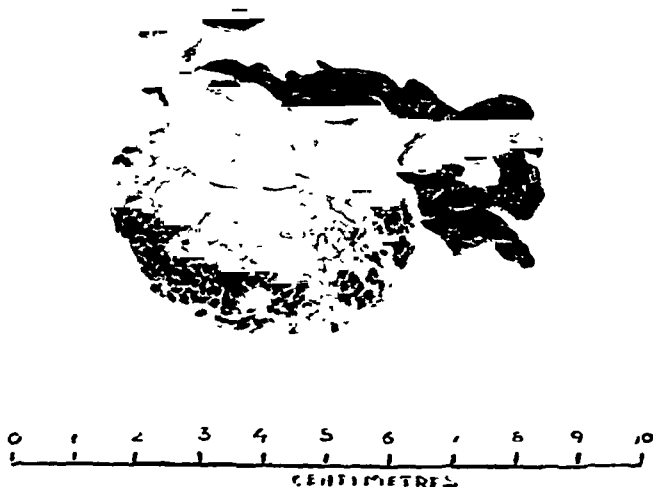


FIG. 388.—Kidney, showing gross reduction in size and the granular pitted surface.

demarcation between cortex and medulla largely lost, and there was considerable increase of pelvic fat. There was no evidence of pelvic or ureteric dilatation, and no pyelitis. The arterial branches and twigs on the cut surface showed no obvious thickening, though the renal artery itself was firmer and more rigid than normal.

Microscopical sections of the kidney present an unusual picture. The general effect (*Fig. 389*) is that of advanced renal sclerosis. Numerous glomeruli are completely obliterated and represented merely by hyaline pink-staining whorls, others are obviously hypertrophied, while still others show varying degrees of atrophic changes and are enclosed in fibrosed thickened laminated Bowman's capsules. The tubules are reduced in number, but greatly dilated, and a moderate number of small cysts are present in the cortex. The tubular epithelium is somewhat flattened. The interstitial tissue is greatly increased in amount, and is widely infiltrated by the usual cells of chronic inflammation, which in places form dense accumulations. The arteries do not show medial fibrosis or hyalinization, nor is there any intimal proliferation. The picture is thus typical neither of chronic nephritis nor of hypertensive nephrosclerosis.



FIG. 389.—Microphotograph showing general renal destruction. The field was chosen to show the shrinking of the glomeruli and the dilated tubules, and represents one of the least affected portions of the kidney. ($\times 60$.)



FIG. 390.—Microphotograph showing one of the larger calcific deposits in the interstitial tissue. In an adjacent degenerated tubule is a crystalline deposit of unknown constitution. ($\times 60$.)

Another unusual feature is the presence of numerous small foci of calcification (*Fig. 390*). These are of varying sizes, the one depicted being one of the largest seen, and occur either in the connective tissue, or within or immediately adjacent to the basement membranes of the tubules. No calcification of glomeruli was noted, nor did there appear to be any calcific accumulations within the tubules, except where occasionally an apparent erosion of the cellular covering to a calcified deposit had left it protruding into the lumen. The assumption that these particles are calcific deposits is based on their staining reaction, their hard and gritty consistency, and the fact that the raised blood-calcium level, the parathyroid hyperplasia, and the experience of other observers, all provide a reasonable excuse for accepting their constitution to be that of salts of calcium.

In addition to these darkly-staining masses another type of foreign body is present in considerable numbers in the cortex. These consist of rounded bodies of peculiar appearance lying apparently within dilated lymphatics (*Fig. 391*). Their appearance is not unlike the conventional picture of the cross-cut of a tree trunk. They resemble, too, the soap plaques seen in fæces, and it is possible that they are indeed calcium soaps formed as an intermediate stage in the process of calcification. This suggestion is supported by the fact that in a few of them appear granules or streaks staining darkly with hæmatoxylin and representing

probably early foci of calcification. The smallest of these bodies appear immediately beneath the tubular basement membrane. Most of them occupy spaces in the newly formed fibrous tissue and are often surrounded by chronic inflammatory cells. They are insoluble in water and in the common fat solvents, and they are not doubly refractile.



FIG. 391.—Microphotograph of a small collection of the interstitial and intralymphatic rounded bodies, which may be calcium-soap plaques. ($\times 60$.)

DISCUSSION

The term renal rickets appears to have been first employed by Barber,⁴ who published a description of ten cases in 1920, but there is no doubt that the association between stunted or deformed growth and chronic renal disease had been recognized long before this. Thus Lucas,⁵ as long ago as 1883, reported a number of cases of rickets associated with albuminuria in late childhood, and used the term 'rickets of adolescence' to describe them. The credit of defining the condition more clearly as a clinical entity probably belongs to Fletcher.⁶ Parsons⁷, Teall⁸, and Brockman²⁴ have added valuable contributions dealing with clinical and radiographic features, while theories on pathogenesis have been elaborated by Parsons⁷ and Mitchell,⁹ and more recently by Shelling and Remsen¹⁰ and by Chown.¹¹

CLINICAL RÉSUMÉ

A brief outline of the more usual clinical features may not be out of place at this stage. Although generally regarded as a disease first manifesting itself in late childhood, it is probably significant that a number of the patients have been reported as being unusually small at birth. The earliest symptoms usually occur about the age of seven, and consist of polyuria and polydipsia associated with a retardation of growth. The characteristic bony changes supervene in later childhood or may be delayed until after puberty, and it is on their account that the child is usually first brought under observation. There may be attacks of renal pain, especially

in those cases associated with secondary pyelonephritis or calculus, but these are by no means common features. The renal lesions, although invariably extensive at post-mortem examination, are, as a general rule, remarkably silent in their course.

The rapid development of genu valgum between the ages of ten and fifteen years is a rather frequent incident, and, occurring in a stunted child with a history of thirst and polyuria, should always suggest the possibility, or even the probability, of renal rickets. Thickening of bony extremities may be present, and subsequently, with increasing softening of the metaphyses, extreme degrees of distortion and deformity may be produced. Displacement of epiphyses is not uncommon, and an abnormal liability to fractures has been reported in several instances.

The majority of cases show little, if any, mental defect, but delayed sex development is common. The skin has a characteristic sallow or earthy tinge, the hair is sparse and dry, and a hypochromic anæmia is the rule. It is noteworthy that cardiac hypertrophy and vascular hypertension are quite exceptional in this disease, notwithstanding the fact that palpable thickening of peripheral vessels is frequently found. Ophthalmoscopic examination is negative in all except the rare cases showing hypertension.

The urine is copious and of persistently low specific gravity. The kidneys are incapable of excreting a concentrated urine. Some degree of albuminuria and cast formation is found in practically all cases. It may be associated in infected cases with pus cells and organisms. Renal function tests usually demonstrate gross inadequacy, and most cases terminate in uræmia.

BLOOD CHEMISTRY

The essential changes comprise azotæmia, lipæmia, and hyperphosphatæmia, usually, but not always, associated with a low calcium figure. There is in addition a lowered alkali reserve, or even, in some cases, an uncompensated acidosis. The blood-phosphatases are increased. The nitrogen retention may reach an extreme degree, a blood-urea or N.P.N. of over 200 mg. per 100 c.c. being quite a common finding.

An amazing feature of this disease is the feeling of comparative well-being which may coexist with such ominously high figures. It is difficult to realize that these patients live constantly beneath the shadow of uræmia, a catastrophe which is liable to supervene with dramatic suddenness, particularly after surgical intervention. In this connection it may not be inapposite to remark that the wise surgeon will set himself steadfastly against any such procedures, however loudly the deformed bones may seem to cry out for osteotomy.

RADIOGRAPHIC APPEARANCES

From a purely radiographic standpoint two quite distinct types of bone change are now recognized :—

Type A.—In this type the shafts of the long bones retain a reasonably normal density, but more or less florid rachitic changes are seen at the extremities differing in no material detail from those seen in ordinary rickets. There is no bending of the shafts, but simple displacement of epiphyses is liable to occur at the softened junctions in situations where they are normally subject to sheering stresses, such as the head of the femur.

Parsons⁷ and Teall⁸ state that it is possible for healing to take place so long as X-ray appearances conform purely to this type, but that healing never occurs in *Type B*.

Type B.—The appearances in this type are unique and quite unlike the picture associated with ordinary rickets. The bones show a general osteoporosis with loss of distinction between cortex and medulla. The cupped metaphysis is both broader and deeper than normal, and shows a ground-glass, woolly, or moth-eaten appearance with varying degrees of stippling. An appearance of subperiosteal erosion may extend for short distances along the diaphysis. Even the flat bones do not escape, and the vault of the skull shows considerable thickening. The osteoporosis here presents a peculiar honeycombed and stippled appearance reminiscent of the acute stage of Paget's disease or of certain cases of multiple osteitis fibrosa. These skeletal changes imply very severe metabolic disturbances, and Teall, who found them present in seven out of his ten cases, regards them as radiographically diagnostic of renal rickets. All the more severe deformities are found in association with this type, which for this reason has been simply termed the 'bending' type. It must be emphasized, however, that it is not so much a bending of the shaft as a collapse of the softened metaphysis which gives rise to the really gross deformities. As Teall puts it, the metaphysis 'collapses sideways', so that the epiphysis may be rotated through 45° or even 90° in a very short time. Secondary epiphysal displacement then occurs very readily, and may add considerably to the deformity.

In our opinion the peculiar radiographic features of *Type B* are probably to be correlated with parathyroid overactivity. It is now known that parathyroid enlargement is found in many of the advanced cases of the disease, and the resemblance to the skeletal changes produced by accepted hyperparathyroidism is too striking to be ignored. The significance of this will be developed in the later discussions on etiology.

The case here reported quite evidently conforms radiographically to *Type B* (Figs. 377-380), and reference has already been made to a considerable enlargement of the parathyroids discovered at autopsy.

REVIEW OF PATHOLOGICAL FEATURES

The pathological features of reported cases fall conveniently into three main divisions, viz., skeletal changes, endocrine abnormalities, and urinary tract lesions. Occasionally lesions occur in other systems, and some of these appear important in particular cases, but in the discussion which follows only the main lesions in these three systems will be considered.

Skeletal Changes.—The naked-eye and radiological appearances of the bones in the great majority of cases are sufficiently similar to those seen in rickets due to avitaminosis D to merit the name of renal rickets. In particular there is the marked enlargement of the epiphyses and the irregularity and broadening of the lines of ossification, and, as a rule, some degree of deformity of the long bones. The resemblance to ordinary rickets is, however, not borne out in all aspects of the bony change; in particular, the microscopical picture of the epiphysal line shows distinctly that the primary osseous and cartilaginous nature of the defective growth in rickets is here replaced by changes which appear to be secondary merely to excessive calcium resorption. While, as in infantile rickets, the line of ossification is irregular and twisted, and the junctional cartilage cells devoid of columnar arrangement,

there is here an absence of the usual rachitic proliferative changes in the cartilage cells. Also the bony trabeculae present have little or no covering layers of osteoid, and the inter-trabecular spaces are filled with fibro-fatty tissue which may even extend into the spaces which should be occupied by marrow.

Shelling and Remsen,¹⁰ Salvesen,¹² and others draw attention to the fact that the histological pictures of infantile rickets and osteomalacia on the one hand, and of hyperparathyroidism on the other, though usually quite distinct, may at times resemble each other to such an extent as to be indistinguishable. The microscopical picture seen in renal rickets is thus of little real value in solving the problem of its etiology, inasmuch as it indicates only that calcium depletion of the skeleton is the essential basis of the changes seen at the lines of ossification, without implicating exclusively either endocrine dysfunction or other disturbances of ossification.

The thickening of the skull seen in most cases is the result of soft and vascular overgrowth of cancellous bony spicules and of intertrabecular fibrous tissue, with loss of differentiation between the diploë and the outer and inner tables.

The Endocrines.—In general it may be said that the thyroid, adrenals, and pancreas show no evidence of abnormalities in these cases, and that the changes in the gonads have not been regarded as primary. The thymus in Shelling and Remsen's¹⁰ case, as in ours, showed diffuse calcification of the Hassall's corpuscles and general atrophic changes; but among the reported cases it has never been considered to play anything more than a passive role. Of the remaining endocrines the parathyroids are so frequently involved as to be regarded by many as being as important as the kidneys themselves. The changes observed are simple hyperplasia of all the glands, or, less frequently, adenomatous enlargement of one or perhaps two of the glands. The histological appearances seen in these hyperplastic glands show no variations from the normal. The proportion of principal and oxyphil cellular elements appears unchanged, and the arrangements of the cells also show no abnormalities.

The pituitary gland has in a few cases been stated to show some minor abnormality of its anterior or posterior portions, but until quite recently it had never been seriously considered as causative of the condition. Chown^{11, 13} has, however, put forward the theory that renal rickets is primarily the result of pituitary dysfunction. The implications of this hypothesis will be considered at a later stage. Meanwhile it may be stated that most of the pituitaries examined have shown little or no pathological change detectable by the means at our disposal.

The Kidneys.—As renal deficiency is the generally accepted basis of the disease entity under consideration, the picture presented is always one of some form of advanced renal destruction. In the majority of cases the kidneys are found to be small, contracted, and scarred, and histologically present the diffuse sclerosis and glomerular obliteration commonly found in cases terminating in uræmia. In a certain number of cases acute infective lesions in the form of a pyelonephritis may be superimposed, rendering difficult a proper appreciation of the pre-existent chronic renal changes. Occasionally the form of renal change is nephrotic in type, as in the case of Kass and Huenekens.¹⁴ Even though in this case death did not result from renal failure the claim to the diagnosis of renal rickets seems well established, and it provides one of the aspects of this clinical entity which does not readily fit into the chemical theory of pathogenesis propounded by Mitchell.⁹

The observations of Ellis and Evans² and of others indicate that in certain cases the renal destruction is consequent upon some abnormality of the lower urinary tract of a congenital nature resulting in lesions such as hydronephrosis or dilatation of the ureters. In a small number of cases the renal lesion found has been congenital cystic kidney.

The microscopical structure seen in the kidneys in the different cases of renal rickets varies as would be expected from the great differences noted in the naked-eye appearances of the kidneys. Essentially progressive and advancing glomerular destruction is the only feature of note in all cases that have run their full course. As a general rule the arterial changes found appear to be secondary in type, and this is in keeping with the rarity of observed hypertension during life. An important feature observed in some cases is the evidence of calcuria provided by the presence of foci of interstitial and occasionally of intra-tubular calcification. Rarely calculi are found. As is to be expected, these cases usually coincide with those in which there is also parathyroid hyperplasia.

ETIOLOGY AND PATHOGENESIS

It has been found an extremely difficult problem to correlate the varied clinical and pathological features with any single etiological factor. One constant finding alone emerges from an examination of the accepted cases, viz., a pronounced degree of renal damage and deficiency in all advanced stages and in all post-mortem examinations. In a high proportion of cases this damage is effected by what Barber describes as "an insidious chronic interstitial nephritis of obscure etiology". In a search for the cause of this obscure form of chronic nephritis specific infections such as syphilis and tuberculosis may be excluded at the outset, as also can be the acute infections of childhood, no one of which occurs with sufficient frequency to warrant its being singled out as possessing causative significance. It is also improbable that any of the ordinary forms of diffuse or focal chronic nephritis are constantly concerned, for in the great majority of these cases there is neither an antecedent history of acute nephritis nor any clinical or post-mortem evidence of hypertension or cardiac hypertrophy. The histological picture presented by the kidneys provides still further evidence that the process is an unusual one. Advanced glomerular destruction and gross tubular dilatation supply the usual evidences of renal failure, but the occasional indications of antecedent acute or subacute toxic nephritis, as provided by thickened and adherent tufts or by crescents in Bowman's capsules, are completely absent, as are also any evidences of a causative hypertensive arteriosclerosis. The only findings indicative of the possible cause of the renal destruction are suggestive of a true chronic interstitial inflammation such as is provided by chronic pyelonephritis. Similar progressive inflammation and fibrosis may conceivably be caused by the presence of any widely diffused chemical or physical irritant in the renal connective tissue. In the case here reported the densely fibrotic kidneys on section showed such a diffuse interstitial irritant in the form of minute calcific deposits. Such calcium deposition has been described in other cases, and attention has been drawn to the fact that analogous changes may occur in primary hyperparathyroidism. Much controversy rages over the possibility of some such purely endocrine causation of the observed renal lesion, and at this stage it may be advantageous to consider the two rival theories of the pathogenesis of renal rickets which at present hold the field. For the want of better names they may be called

the 'renal' and the 'endocrine' theories of origin, and the arguments which have been marshalled in support of each may now be considered.

The Renal Theory.—The renal theory of origin, briefly expressed, states that as the result of chronic renal disease in childhood there develops a condition of rickets with the attendant skeletal changes and deformities, and usually also with some degree of dwarfism and possibly infantilism. That renal deficiency usually terminating in uræmia is a more or less constant feature of the clinical syndrome has already been stressed, and it remains therefore to explain the manner in which the renal damage is believed to produce the skeletal changes. There are several modifications of the basic theory, but they all start with the inability of a damaged kidney to effect an adequate excretion of phosphates. The immediate result of this is a tendency to a raised phosphate level in the blood. The serum values of calcium and phosphorus are more or less complementary, and Parsons,⁷ to whom the earlier conceptions of this theory are due, pointed out that by reason of the raised value of blood-phosphates a relative if not an actual deficiency of blood-calcium resulted. This relative calcium deficiency interferes with the deposition of bone, and results in an endogenous form of rickets.

Mitchell,⁹ starting with the same premises, has considerably modified these views, and produced a theory which is the one generally accepted by the protagonists of the 'renal' hypothesis. According to his views, the fact that the unexcreted phosphates do not bank up progressively in the blood is explained by the fact that an alternative route of excretion is provided by the bowel. Mitchell believes that in the gut the excreted phosphorus may reach such concentrations as seriously to interfere with the absorption of the food calcium. Experimental evidence has been adduced in support of this theory. Thus rickets is readily produced in rats by a diet containing either high-calcium and low-phosphorus, or low-calcium and high-phosphorus, contents. It would appear that an excess of either one interferes with the absorption of the other. Similarly Orr, Holt, Wilkins, and Boone¹⁵ have shown that in human infants excessive intake of either calcium or phosphorus interferes with the absorption of that element which happens to be in the lower concentration. Mitchell suggests that the excess phosphorus accumulating in the intestine by excretion acts in an identical way, fixes the food calcium as an insoluble calcium phosphate, and so leads to a true calcium deficiency rickets.

Theoretically such a calcium deficiency would appear to call for intensive administration of calcium by mouth in order to counteract any such discrepancy between calcium and phosphorus concentrations. In this connection it is of interest to note that Salvesen¹² claims to have benefited one of his cases materially by giving large doses of calcium lactate by mouth. The failure of ultra-violet light and of vitamin D concentrates to cure renal rickets also finds an explanation on Mitchell's theory, for as the calcium is already fixed no amount of vitamin will enable it to be absorbed.

These arguments afford a reasonable explanation of the bone lesions so long as a hypocalcæmia is present; but, as has already been noted, cases showing a normal or raised serum calcium are by no means rare. Figures as high as 15 or 16 mg. per cent have been obtained on repeated estimation in undeniable cases, and it is to be presumed that such hypercalcæmia is related to an increased activity if not to an actual hyperplasia of the parathyroid glands, particularly so as among the most recently investigated cases an increasing proportion of them do show

enlargement of some or all of the parathyroid glands. This hyperparathyroidism must be explained, and several suggestions have been put forward in this connection. One of the earliest of these presumes that a mobilization of calcium from the bones under the influence of the parathyroids is a necessary expedient to prevent the onset of tetany; but it is probable that this is not the whole truth. For one thing tetany is strangely uncommon in renal rickets, despite a calcium figure which sometimes falls as low as 4 mg. per cent. Actually a defence against tetany already exists in the form of an increased acidity of the blood. It is now recognized that calcium exists in the blood in two main forms, 'free' and 'bound', the latter being in loose combination with the plasma proteins. The ionization of the 'free' calcium depends on the pH of the plasma; the more this is lowered to the acid side, the greater the number of free calcium ions. The incidence of tetany appears to correspond rather closely with the reduction in the amount of ionized calcium present. In this connection it is interesting to note that by a method of ultra-filtration through collodion sacs, Pincus, Peterson, and Kramer¹⁶ have shown that in chronic renal disease, although the total calcium in the blood may be much reduced, the reduction occurs mainly at the expense of the bound calcium, the free form being very little lowered. It may be presumed that a similar relationship between free and bound calcium occurs in renal rickets, and, as in addition the pH value of the blood often reveals an uncompensated acidosis, the comparative rarity of tetany, even when the total calcium is low, is easier to understand. On the other hand, the necessity for parathyroid hyperplasia becomes correspondingly more difficult to explain, especially when it is of sufficient degree to raise the calcium level to the high figures occasionally found. Actually it seems probable that too much attention has been focused on the blood-calcium, and that a more reasonable explanation is to be found in the hyperphosphatæmia. Admittedly hyperparathyroidism tends to correct a low calcium, but it also tends to correct a high phosphate level, and, as we have already shown, it is this retention of phosphorus which is mainly responsible for the metabolic embarrassment. Ellsworth and Howard¹⁷ and also Shelling¹⁸ have shown that administration of parathyroid principle causes mobilization and increased excretion of phosphate in man and animals, and it is probably to this end that the parathyroids undergo hypertrophy. On this view the hypercalcæmia is to be regarded as an almost accidental accompaniment, although admittedly unfortunate in the serious effect on skeletal stability with which it is associated.

Before leaving the renal theory it is to be noted that there are certain features of the syndrome of renal rickets which do not find a readily acceptable explanation on the basis of its arguments. It is pointed out by the opponents that the dwarfism, with or without infantilism, which is usually present, cannot easily be accounted for by chronic renal disease, particularly as these features are usually manifested in renal rickets long before the renal condition has progressed to the advanced stage at which they are likely to appear. On the other hand, these same features are very readily explicable on the basis of a primarily endocrine disturbance.

The Endocrine Theory.—The endocrine theory presumes a primary over-activity of the parathyroid glands, with or without an over-riding pituitary dysfunction. The former is sufficient to account for the skeletal demineralization and the deformities, but the dwarfism and infantilism are more obviously the effects of the pituitary. If it be admitted that the parathyroids are under pituitary control the problem is further simplified, for the whole of the mischief is then attributable

to the misdirected energies of this arch-dictator of the endocrines. The renal changes according to this theory are of a secondary nature. It is assumed that as a result of the hyperparathyroidic hypercalcaemia the kidneys will excrete calcium in excess. That calcuria does supervene in cases of hyperparathyroidism is proved, and it is suggested that in the process of calcium excretion some of the calcium reabsorbed from the tubules will accumulate in the peritubular lymphatics and tissue spaces, and there set up chronic simple irritative inflammation. This form of true interstitial chronic nephritis can be seen around calcific deposits in cases of osteitis fibrosa, and in many cases of renal rickets, including our own, a similar condition has been observed. That this interstitial inflammation can go on to destruction of the renal parenchyma seems undoubted, and thus the renal deficiency is fully accounted for as a secondary result of hyperparathyroidism.

Enlarged parathyroid glands have been reported in many of the recently recorded cases. Thus Shelling and Remsen reported hyperplastic enlargement of all four glands in their case, and they actually demonstrated, using Hamilton's¹⁹ test, an increased content of parathormone in the patient's blood. Other cases, in addition to our own, have been reported by Smyth and Goldman,²⁰ Langmead and Orr,²¹ and by Pappenheimer and Wilens.²² That the calcific renal deposits found in these cases can account for the renal sclerosis usually present at post-mortem is suggested by some experimental observations. From the work of Albright²³ and others it is evident that extreme degrees of damage to the kidneys may result from the prolonged action of excessive parathyroid principle. In the experimental animal it is even possible by the administration of large doses of parathormone to cause complete anuria and death from renal failure in a few days.

Some of the arguments brought against this theory merit discussion. It is maintained that hyperparathyroidic skeletal demineralization produces a different radiographic picture from that of renal rickets. This is true of *Type A* renal rickets, but in the commoner *Type B* the pictures are often indistinguishable. In this connection it is noteworthy that *reported* cases of osteitis fibrosa in childhood are extremely rare. We would suggest that this may result from the fact that the skeletal deformities which develop in the growing bones resemble rachitic lesions so strongly that clinical attention is apt to be directed away from the real etiology of the condition, and that in fact these cases have been diagnosed as renal rickets. Another criticism levelled against the endocrine theory is that in large numbers of the reported cases there is no mention of parathyroid enlargement and/or calcific deposits in the kidney. That this is true of the great majority of the earlier reported cases is conceded; but examination of recent reports shows that these features appear in a large proportion of the cases in which search was made for them. The absence of renal calcific deposits may actually have little significance, as by the time the kidneys are examined they represent merely the burnt-out hulks in which a search for the cause of the fire which destroyed them can have little reasonable hope of success. That the renal calcific deposits are not present in much larger amounts is almost certainly due to the fact that the process of inflammation set up by them inevitably results in their partial or total removal.

A more serious objection to the endocrine theory is provided by the group of cases, already referred to, in which the renal damage is associated with congenital abnormalities of the urinary tract. Ellis and Evans,² describing twenty cases of

renal rickets, report the presence in fourteen of them of ureteric dilatations which are believed to be idiopathic and probably the result of a type of congenital neuromuscular imbalance at the ureteric orifices allied to the achaliasias. Mitchell, in his large survey of cases, refers to a group of nine or ten in which renal damage was the result of congenital cystic kidneys. In other cases there have been other congenital lesions such as horseshoe kidney, ureteric stenosis, solitary kidney, etc. Such a high incidence of cases of these types does not accord readily with the endocrine theory, particularly when the congenital lesion is obviously sufficient in itself to account for the renal damage present. It may, however, be pointed out that, to those who accept the pituitary dysfunction theory wholeheartedly, such features as congenital abnormalities of the urinary tract find a rational explanation in an assumed disturbance in the adjacent diencephalic ganglia which are believed to control the development of the renal system.

Conclusions.—Such, then, are the two main theories and the arguments for and against each. It will be noted that each hypothesis is capable, with a little stretching, of accounting for most, if not all, of the features of renal rickets, and it behoves us to declare ourselves in favour of one or other of the two conflicting theories. Before attempting this it is essential that we define the syndrome of renal rickets. If we accept the definition of Shelling and Remsen, viz., “rickets resulting from chronic renal disease”, then obviously there is no argument as to which of the two theories is to be accepted. In fairness to these authors it should be pointed out that their definition, which so finally excludes any discussion as to the etiological nature of the disease, was made in the full acceptance of Mitchell's theory, and for the purpose of excluding cases of diverse types which had at one stage or another been included under the heading of renal rickets.

In our opinion the only possible definition of renal rickets at the present stage of our knowledge is one which makes no mention of etiology, but merely expresses the relationship of the principal features, e.g., “a disease of childhood characterized by skeletal demineralization with resultant deformities, and associated with chronic renal disease which in uncomplicated cases terminates in uræmia”. The acceptance of such a definition inevitably admits under the banner of renal rickets many cases which protagonists of the purely renal theory of origin would exclude, and raises the question whether the cases falling under this title do not perhaps belong to two, or even more, distinct disease entities. It must be clearly appreciated that the diagnosis of renal rickets is *not* made until both skeletal and advanced renal changes are present. It appears to us probable that the clinical condition diagnosable as renal rickets may be, not a disease entity of itself, but a syndrome common to either of two separate and distinct diseases in their late stages. Our discussion of the two theories of etiology of this syndrome has thus been directed towards explaining the pathogenesis of the two separate diseases, each capable of evolving a late phase which has been designated ‘renal rickets’. The retention of the name ‘renal rickets’ for this syndrome is in our opinion desirable, as not only is the condition a distinctive one, but the alternatives of classifying all our accepted cases as merely ‘late sequelæ’ of hyperparathyroidism on the one hand, or of renal deficiency on the other, is not attractive. Justifiable diagnoses of renal rickets will therefore continue to be made, and although in many there will be little or no indication as to the parent condition from which they arose, there are nevertheless certain diagnostic ‘pointers’ which may be of assistance.

At one extreme there is that group in which the primary disorder is endocrine. For our purpose it matters little whether we assign the major role to the parathyroids or to the pituitary. The skeletal changes are in any case to be regarded as the immediate result of a parathyroid overactivity, the effect on the sensitive growing metaphyses being especially severe, and producing a condition which in children may be indistinguishable from rickets. The hypercalcaemia which results from the extensive demineralization of the bones causes in its turn a progressive nephrosclerosis, terminating ultimately in renal failure. Clinically some degree of demonstrable bony change precedes the evidences of renal disease, so that any marked degrees of nitrogenous and phosphatic retention are late features. Collateral evidences of pituitary or diencephalic disturbances may be present, and in conjunction with the antecedent history may assist in the differentiation from the primarily renal group.

At the other extreme we have those cases arising as a result of congenital or other disturbances in the anatomy or physiology of the renal tract. Here the skeletal changes are secondary to the renal disease, and are to be regarded as the result of calcium deprivation, probably for the reasons suggested by Mitchell. Evidences of renal impairment appear early, and are associated with definite nitrogenous retention and with hyperphosphataemia, while the blood-calcium remains low. When the metabolic disturbances reach a critical level, secondary parathyroid hyperplasia is evoked, and with the advent of this parathyroid overactivity the skeletal manifestations begin to alter. A progressive decalcification causes a general softening of the bones, and precipitates the characteristic metaphysial collapse which we know to play so important a part in producing many of the more grotesque deformities. Radiographically the picture which previously conformed to that of *Type A* renal rickets now takes on an unmistakable parathyroid imprint, and gives rise to the appearances recognized as *Type B*. Simultaneously there is a rise in the blood-calcium above normal levels, and secondary calcific deposits may form in the kidneys, blood-vessels, and elsewhere. From this point the onset of terminal renal failure is only a question of time. If it is known that albuminuria, cylindruria, or other evidences of renal trouble preceded clinical or X-ray signs of bone disease, and are associated with relatively low serum calcium and high phosphorus figures, the implications are that the case belongs to the renal group. Confirmatory evidence to the same effect will also be afforded if it is possible to demonstrate bilateral dilatation of the urinary tract or other congenital renal abnormalities by means of excretory urography.

It will be evident that with the appearance of severe renal damage in the first group and of secondary parathyroid hyperplasia in the second, a zone of overlap between two etiologically distinct diseases has been reached. It is here that the two pathways converge, and it is here also that the diagnosis of renal rickets is most frequently made. In many cases it will be extremely difficult to retrace with any certainty the steps by which this common ground has been reached. Such a difficulty confronted us in the case recorded in this paper. The hypercalcaemia and radiographic features pointed to hyperparathyroidism. At the same time there was such a degree of renal impairment that uraemia could not be long delayed. Little assistance was afforded by the history or by other clinical features, with the possible exception of the unusually long-standing polyuria and the fact that the dwarfism appeared to date from birth. These facts may be adduced as evidence

in favour of some form of pituitary or diencephalic disturbance, and the absence of any congenital renal lesion in the post-mortem findings (with due regard to their limitations) may be presumed to lend further support to this view. With these reservations we therefore conclude that our case should properly be assigned to the 'endocrine' category.

SUMMARY

A case of renal rickets is described, with illustrations. The literature dealing with this condition is reviewed, and the clinical, biochemical, radiographical, and pathological features are summarized.

The 'renal' and the 'endocrine' theories of origin of the condition are critically discussed. The concept of renal rickets as a syndrome representing the common ground of overlap of hyperparathyroidism on the one hand, and of chronic renal damage on the other, is expounded and suggested as the most satisfactory in the present state of our knowledge.

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REFERENCES

- ¹ PARSONS, L. G., *Arch. of Dis. in Childh.*, 1927, ii, 198.
- ² ELLIS, A., and EVANS, H., *Quart. Jour. Med.*, 1933, ii, 231.
- ³ BRAILSFORD, J. F., *The Radiology of Bones and Joints*, 2nd edition, 1935, 484. London: J. & A. Churchill.
- ⁴ BARBER, H., *Quart. Jour. Med.*, 1921, xiv, 205.
- ⁵ LUCAS, R. C., *Lancet*, 1883, i, 993.
- ⁶ FLETCHER, H. M., *Proc. Roy. Soc. Med. (Sect. Dis. Child.)*, 1911, 95.
- ⁷ PARSONS, L. G., *Arch. of Dis. in Childh.*, 1927, ii, 1.
- ⁸ TEALL, C. G., *Brit. Jour. Radiol.*, 1928, xxi, 25.
- ⁹ MITCHELL, A. G., *Amer. Jour. Dis. Child.*, 1930, xl, 101, 345.
- ¹⁰ SHELLING, D. H., and REMSEN, D., *Bull. Johns Hopkins Hosp.*, 1935, lvii, 3, 158.
- ¹¹ CHOWN, B., *Brit. Jour. Surg.*, 1936, xxiii, 552.
- ¹² SALVESEN, H. A., *Act. med. Scand.*, 1934, lxxxiii, 485.
- ¹³ CHOWN, B., *Canad. Med. Assoc. Jour.*, 1936, Aug., 134.
- ¹⁴ KASS, I. H., and HUENKENS, E. J., *Ohio State Med. Jour.*, 1935, xxxi, 119.
- ¹⁵ ORR, W. J., HOLT, L. E., jun., WILKINS, L., and BOONE, F. H., *Amer. Jour. Dis. Child.*, 1924, xxviii, 574.
- ¹⁶ PINCUS, J. B., PETERSON, H. A., and KRAMER, B., *Jour. of Biol. Chem.*, 1926, xlviii, 601.
- ¹⁷ ELLSWORTH, R., and HOWARD, J. E., *Bull. Johns Hopkins Hosp.*, 1934, lv, 296.
- ¹⁸ SHELLING, D. H., *The Parathyroids in Health and Disease*, 1935. St. Louis: C. V. Mosby Co.
- ¹⁹ HAMILTON, B., and SCHWARTZ, C., *Jour. Pharmacol. and Exper. Therap.*, 1932, xlvi, 285.
- ²⁰ SMYTH, F. S., and GOLDMAN, L., *Amer. Jour. Dis. Child.*, 1934, xlviii, 596.
- ²¹ LANGMEAD, F. S., and ORR, J. W., *Arch. of Dis. in Childh.*, 1933, viii, 265.
- ²² PAPPENHEIMER, A. M., and WILENS, S., *Amer. Jour. Pathol.*, 1935, xi, 73.
- ²³ ALBRIGHT and others, *Arch. of Internal Med.*, 1934, liv, 315.
- ²⁴ BROCKMAN, E. P., *Brit. Jour. Surg.*, 1926-27, xiv, 634.

CHOLESTEROSIS OF THE GALL-BLADDER: A REVIEW, SUPPLEMENTED BY PERSONAL OBSERVATIONS ON 87 CASES

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INTRODUCTION

THE term 'cholesterosis' of the gall-bladder designates an interesting and fairly common condition in which the mucous membrane is infiltrated with a mixture of cholesterol esters and neutral fat. This lipid material is distributed in a patchy fashion, forming bright yellow flecks of variable size, sometimes slender and scanty, sometimes distending each mucosal fold so that the gall-bladder seems to be lined with a thick, soft, golden, waxy fabric. The strands of this fabric run longitudinally, and terminate a short distance from the neck of the gall-bladder. Occasionally cholesterol polyps of mulberry form are present, and in some gall-bladders such are the sole site of gross lipid deposit. The subserous coat contains an unusual amount of ordinary fat (*see Fig. 403*), and this fact may encourage the surgeon to essay a diagnosis of cholesterosis before the gall-bladder is opened. An excess of fat, however, frequently occurs in this site apart from cholesterosis. Gall-stones are found in about a third of all cases and are almost invariably of cholesterol-rich types. (*Figs. 392, 393.*)

The lipid infiltration is finely dispersed within cells, both of the lining epithelium and of the supporting connective tissue, forming a sort of emulsion in the cytoplasm and producing a characteristic foamy microscopic picture. In the epithelium the lipid may consist merely of a few globules in the basal parts of the cells, or, in extreme cases, the cytoplasm may be distended with droplets of varying sizes. The globules tend to be arranged in rows perpendicular to the free surface of the cells, and to be graduated so that the largest are next to the basement membrane (Policard, 1914). In the stroma the lipid is contained in macrophages, which aggregate especially in the swollen tips of the plicae. These aggregations may assume such proportions as to form the polypoidal masses mentioned above.

The story of cholesterosis is of recent growth, and the first reference to a condition of this nature was in 1857, when Virchow described a type of fatty infiltration of the mucosa of the gall-bladder, although he regarded the deposit as neutral fat excreted by the liver and reabsorbed by the gall-bladder. Aschoff (1906) observed the occurrence of cholesterol in the epithelium of the gall-bladder. Attention was directed afresh by Moynihan (1909) to yellow stippling of the mucosa, and it assumed a new importance owing to his terming it "a condition of the gall-bladder requiring cholecystectomy". Thereafter cholesterosis attracted the attention of surgeons and pathologists who previously had overlooked the condition, or who, like Virchow and Aschoff, had not regarded it as abnormal. Moynihan

was misled, however, regarding the nature of the lesion, for he believed the yellow flecks to be due to bile-staining of the submucous fibrous tissue as a result of desquamation of the epithelium from the tips of the plicæ. This error was repeated by MacCarty (1910), who coined for one type of the condition the now widely used name 'strawberry gall-bladder'. This attractive metaphor is apposite only to strawberries not fully ripe, but it is so picturesque that it has been translated into many languages. It has led numerous writers to suppose mistakenly that in cholesterosis the general background of the mucous membrane is dark red, and thence to make the



FIG 392—Cholesterosis of the gall-bladder. The plicæ of the mucosa are richly infiltrated with myelin fat, disposed as tortuous brightly-shining columns, in a general longitudinal direction. (Natural size)

specious deduction that cholesterosis is in essence a type of inflammation. Laroche and Flandin (1912) noted the association of cholesterosis with cholelithiasis. Lichtwitz (1914) went further and held cholesterosis to be an important contributing cause of cholelithiasis, for he believed that lipoid polypi, shed into the lumen, provide nuclei for gall-stone formation. This theory has received the support of many later writers (e.g., Gosset, Bertrand, and Loewy, 1928).

From the histological appearances of the gall-bladder epithelium Policard (1914) deduced that cholesterosis is due to resorption of cholesterol from the bile.

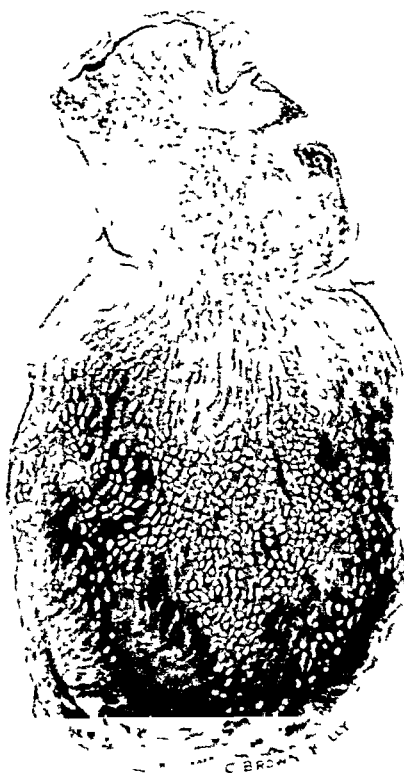


FIG 393—Cholesterosis of the gall-bladder with cholesterol-rich stones. The lipoid infiltration of the mucosa is abundant and characteristic. In this case the stones were recovered from the common bile-duct, having escaped down the wide cystic duct. The largest stone is a 'cholesterol solitaire', the smaller ones are of mulberry type. Note the pigment centre of the split mulberry stone.

Boyd (1922 and 1923) showed that in cholesterosis the dried mucosa of the gall-bladder may contain as much as 60 per cent by weight of lipid as against 0.6 per cent in normal controls, thus proving the condition to be a true infiltration. He showed also that the fatty material in the lipid gall-bladder consists of cholesterol esters, and he was able to demonstrate these in practically all the tissues of the wall. Moreover, he found microscopic lipid in about half of all gall-bladders examined. In the cholesterol gall-bladder he invariably found low-grade inflammatory changes, and concluded that these are the most important etiological factor, and Illingworth (1929), from an extensive pathological and clinical study, endorsed this view in the main, while pointing out that in some cases of cholesterosis evidence of inflammation is hard to find. Mentzer (1925) recorded some degree of cholesterosis of the gall-bladder in 37 per cent of all cases coming to autopsy at the Mayo Clinic. These figures and those of Boyd emphasize how extremely common is lipid infiltration of the mucosa of the gall-bladder, but it is certain that in a large proportion of Mentzer's cases the amount of lipid was quite small. Despite these observations indicating the extremely high incidence of some degree of cholesterosis, it has generally been regarded as a definite type of cholecystitis. Corkery (1922) is practically alone among later writers in suggesting that cholesterosis may not be a specific lesion but merely a random element in the protean manifestations of chronic cholecystitis.

It is commonly believed that the lipid material is derived from the bile, from which cholesterol is resorbed by the activity of the lining epithelium of the gall-bladder. Elman, Graham, and Taussig (1931 and 1932), on the other hand, maintain that the gall-bladder secretes cholesterol, and that cholesterosis results when this secretory mechanism is impeded by low-grade inflammatory processes.

Despite much study and prolific writing many questions remain unanswered with regard to cholesterosis. Whence comes the lipid—from the blood or from the bile? Is cholesterosis a manifestation of infective cholecystitis? Does it lead directly or indirectly to gall-stone formation? Does it of itself produce symptoms? It is the object of this paper to attempt to shed some additional light upon these problems, which will be discussed as indicated in the following synopsis:—

1. THE SOURCE OF THE LIPOID IN CHOLESTEROSIS.

- a. Experimental work on the cholesterol function of the gall-bladder.
- b. The biliary cholesterol in human cases.
- c. Resorption of substances other than cholesterol.
- d. Blood cholesterol in man and animals.
- e. Histological evidence regarding the cholesterol function of the gall-bladder.

2. EXPERIMENTAL PRODUCTION OF CHOLESTEROSIS.

3. IS CHOLESTEROSIS A SPECIFIC FORM OF CHOLECYSTITIS?

4. GALL-STONE FORMATION IN CHOLESTEROSIS.

5. BACTERIOLOGY OF CHOLESTEROSIS.

6. CHOLECYSTOGRAPHY IN CHOLESTEROSIS.

7. CLINICAL CONSIDERATIONS.

1. THE SOURCE OF THE LIPOID IN CHOLESTEROSIS

Cholesterosis of the gall-bladder is essentially a form of xanthoma, arising as a result of the storage of a local surfeit of lipid. This lipid may be derived either from the blood or from the bile. If the source is the blood, then some

explanation must be sought for the great frequency of cholesterosis (*see, e.g., Mentzer*): why should the gall-bladder act as a magnet, so to speak, for cholesterol? Is the cholesterosis a manifestation of a cholesterol-excreting function normal to the gall-bladder? On the other hand, if the source be the bile, then the gall-bladder must be capable of resorbing cholesterol from the bile. In order, therefore, to clarify the question of the pathogenesis of cholesterosis it is necessary to solve the cognate question of the cholesterol function of the gall-bladder. There are three possibilities: the gall-bladder may secrete cholesterol; it may resorb it; or it may pursue the middle path of indifference. The problem may be attacked (a) experimentally, (b) by study of human cases.

a. Experimental Work upon the Cholesterol Function of the Gall-bladder.—Aschoff (*loc. cit.*), interested in cholesterol metabolism because of its bearing upon the formation of gall-stones, showed in a series of experiments on dogs that the gall-bladder mucosa frequently contains cholesterol esters histologically demonstrable. If, however, the cystic duct had been ligated beforehand, these esters could not be demonstrated, and so he concluded that the lipoid in the epithelium of the gall-bladder mucosa had its origin in resorption from the bile. He was unable directly to prove resorption of cholesterol, although he showed the resorption of neutral fats. He suggested, therefore, that normally the gall-bladder must resorb the cholesterol esters from the bile, and split these, returning the cholesterol fraction to the lumen. Blaisdell and Chandler (1927), by means of a diet rich in lipoids, were able to produce gross deposits of cholesterol in the gall-bladders of all of 10 dogs and 4 of 15 rabbits. Since ligation of the cystic duct prevented the appearance of these deposits, they concluded that the latter were the result of resorption from the bile. Torinoumi's (1923) experiments suggest that the normal gall-bladder of the dog takes up cholesterol from the bile, but if the gall-bladder be the seat of inflammation, either as an accidental result of operative interference, or intentionally from the introduction of typhoid bacilli, cholesterol will be poured out into the lumen, presumably with the exuded serum. Unfortunately these conclusions were based upon only three normal and two inflamed cases. Kusnetowsky's (1923) experiments dealt mainly with the large ducts, but also with the gall-bladder. It was shown that these structures readily took up fat and cholesterol esters introduced into their lumina. Illingworth (1929) demonstrated in the cat the disappearance of cholesterol from the lumen of the gall-bladder segregated by ligation of the cystic duct. He failed, however, to demonstrate it histologically in the wall of the gall-bladder, and suggested that in the gall-bladder, as probably also in the intestine, the cholesterol is taken up in a masked form which is not readily demonstrable with the polariscope. Loewy (1932) also concluded that the gall-bladder resorbs cholesterol. The most recent work on the cholesterol function of the gall-bladder is by Rousselot and Bauman (1935). These workers appear to have demonstrated without much doubt that the gall-bladder is capable of taking up cholesterol from a watery solution with bile-salts, and converting it into cholesterol esters.

Careful consideration must, however, be given to work published in recent years by Elman and Taussig (1931) and Elman and Graham (1932) suggesting that a function of the gall-bladder may be the excretion of cholesterol, i.e., directly the reverse of that mentioned above. Bile was subjected for varying periods to the activity of the mucosa of the gall-bladder which had been isolated by ligation of

the cystic duct. The amount of simple concentration that occurred was measured by comparing the concentration of bilirubin in specimens obtained by puncture at the beginning and at the end of the experiment. The concentration of cholesterol in each of these specimens was also determined. It was found that the cholesterol concentration increased more rapidly than the bilirubin concentration. Elman and Graham maintain that "since in these experiments bilirubin is indifferent to secretion or resorption by the biliary mucosa, its measurement furnishes a convenient gauge of changes in volume due to the action of the gall-bladder on the bile". If this premise be accepted, it is clear that increase of the cholesterol-bilirubin ratio indicates secretion of cholesterol, decrease of the ratio its resorption. The question, however, of the resorption or otherwise of bilirubin is one of the crucial questions upon which depends the correctness of the interpretation of these and many other experiments. It cannot be summarily dismissed in this fashion and it will be more fully discussed below.

More convincing evidence that cholesterol may actually be added to the bile during its sojourn in the gall-bladder is furnished by certain further experiments of Elman and Graham. The arrangement of the hepatic lobes and ducts in the dog renders it possible by appropriate ligations and cannulations to separate the biliary system into two districts, one of which (the central hepatic duct system) communicates with the gall-bladder. The bile from these two districts may then be collected separately, that from the central zone alone having been subjected to the activity of the gall-bladder mucosa. In the experiments under consideration the amount of liver tissue secreting into each duct system was determined approximately. In the specimens from each district an estimation was made of the amount of cholesterol present per gramme of liver tissue. It was found that in the specimen which had been subjected to the action of the gall-bladder, this was about twice as great as in the specimen coming directly from the liver. The authors concluded that cholesterol had been added in the gall-bladder.

These conclusions regarding the cholesterol function of the gall-bladder must, however, be scrutinized in the light of certain errors which are likely to upset the accuracy of such experimental work on the biliary tract. These are: (1) Bilirubin estimations are not a satisfactory control for cholesterol, since, as will be shown, bilirubin may be resorbed from the gall-bladder. (2) In experimental animals there frequently occur accessory bile-ducts, discharging directly into the gall-bladder, and through these cholesterol may be added to the contents of the latter despite ligation of the cystic duct. (3) As a result of trauma or inflammation, large amounts of cholesterol may be added to the contents of the gall-bladder by exudation of blood-serum.

The Inaccuracy of Bilirubin Estimations as an Index of Water-resorption Occurring in the Gall-bladder.—That the gall-bladder may not always be 'indifferent' to bilirubin is shown by the fact that even in the experiments of Elman and Graham the total amount of bilirubin definitely decreased as the experiments proceeded, and at the end of fifteen days it had disappeared. The brilliant work of Rous and McMaster (1921), which directed renewed general attention to the concentrating activity of the gall-bladder, also furnishes on re-analysis strong evidence of resorption of bile-pigment. It was shown in a series of dogs that when the gall-bladder is in communication with a system of ducts, from which discharge of bile is prevented by ligature, the gall-bladder and the ducts soon become filled with

dark bile thickened by addition of mucus. The highest concentrations of pigment were obtained, however, in experiments of quite brief duration, and in experiments lasting many days the amount of pigment in the ducts and also in the gall-bladder was considerably less. It may be argued that since the concentration effected by the gall-bladder is by a process of resorption of water and crystalloids which maintains the osmotic pressure of the gall-bladder bile equal to that of duct bile and also blood-plasma (Brand, 1902), the gall-bladder ceases to exercise an effective concentrating power as soon as the osmotic pressure of the less readily absorbed constituents of the contained bile approximates to that of the plasma. The fact, however, that the pigment content of the duct bile does not rapidly fall indicates that an important proportion of the concentrating activity of the gall-bladder is continuing; under these circumstances actual diminution in the pigment content of the gall-bladder surely strongly suggests resorption. Rous and McMaster note that disappearance of pigment may be masked for a time by conversion of bilirubin into the more deeply coloured biliverdin.

In certain of the experiments the biliary system of the dogs was separated by means of partitioning ligatures into a central area communicating with the gall-bladder, and a peripheral with the distal portion of the common bile-duct. So great was the concentration effected by the gall-bladder that, having to deal with only a fraction of the total bile secretion, it tended to collapse, and came to contain a small amount of dark viscid bile. This frequently contained less pigment than was to be expected from the area of liver drained by the central duct system, *but the total output of pigment remained constant*. Rous and McMaster suggest a selective inhibition of secretion in the central area of the liver, but it seems equally reasonable to assume that pigment was resorbed by the gall-bladder and re-excreted by the liver. Especially so since Aschoff (1909) demonstrated histologically resorption of bile-pigment by the epithelium of the gall-bladder. Further proof of resorption of bile-pigment by the gall-bladder is furnished by the absence of pigment from the contents of the hydropic gall-bladder encountered clinically. The gall-bladder becomes progressively distended with mucoid fluid and the pigment disappears. Graham has suggested that in some instances disappearance of bile-pigment from the gall-bladder may be the result of bacterial action, but in simple hydrops this may probably be excluded.

It is well known, of course, that in operative procedures upon the biliary tract of dogs a complicating infection is extremely difficult to avoid, and Riegel, Johnston, and Ravdin (1932) have shown by quantitative experiments that in the presence of infection 36 per cent of the bile-pigment may disappear from the gall-bladder in as short a time as two hours.

In view of all these facts, the conclusion is inevitable that, in animal experiments, repeated colorimetric estimations of bilirubin in the gall-bladder bile are quite unreliable as a standard whereby to judge the amount of concentration undergone by other substances in the bile. In other words, repeated calculations of the cholesterol-bilirubin ratio will not furnish accurate information as to whether cholesterol is being added to or taken up from the bile. If bilirubin is resorbed, then unless cholesterol be taken up at an equal or greater rate, the cholesterol-bilirubin ratio will inevitably rise. It is probable that the degree of water-resorption will be considerably underestimated; if this be so, the results of such calculations may fallaciously suggest that cholesterol has actually been added

to the contents of the gall-bladder. It is clear that the problem is capable of solution only by definitely quantitative experiments.

Is it Possible for Cholesterol to be Added to the Contents of the Gall-bladder Independently of Secretory Activity on the Part of the Mucosa?—Recent work of Andrews, Dostal, and Hrdina (1933) drew attention to the frequently ignored fact that the blood-plasma normally contains much more cholesterol than the hepatic or even the cystic bile. The figures vary, but the plasma cholesterol of the dog and of man is roughly 200 mg. per 100 c.c., while the gall-bladder bile normally contains 50–150 mg. per 100 c.c. Accordingly, in any experiments upon the cholesterol function of the normal gall-bladder, it is necessary to exclude the possibility of the exudation of serum into the lumen. In only one of Elman's experiments was the final concentration of cholesterol in the gall-bladder (271 mg. per 100 c.c.) greater than the accepted normal value of the plasma cholesterol in the dog, and in the remainder it was much lower. Any increase of total cholesterol, if such there were, might be explained as due to out-pouring of serum as a result of the trauma of operation. The highest reading could have been attained as the result of serous exudation supplemented by a slight exercise of the concentrating function of the gall-bladder.

Wilkie and Doubilet (1933) showed by experiments upon dogs that if the cystic duct be ligated, cholesterol may be added to the bile in the gall-bladder, or withdrawn from it, according as the concentration of cholesterol in the gall-bladder is less or greater than that in the blood-plasma. This implied in most cases a substantial addition of cholesterol to the gall-bladder bile. If, however, the cholesterol content of the gall-bladder bile had been artificially raised by direct addition of cholesterol to a value greater than the concentration in the plasma, then cholesterol was resorbed. In other words, in respect at least of its richness in cholesterol, the fluid in the gall-bladder came to resemble more closely the blood-plasma, the gall-bladder wall behaving as a membrane permeable to cholesterol. Wilkie and Doubilet state, however, that they were compelled to discard the majority of their animals because of inflammatory or gangrenous changes arising as the result of ligation of the cystic duct. The normality of the remainder is not therefore above suspicion, and so while these experiments are interesting as demonstrating the permeability of the inflamed mucosa, they cannot be accepted without reservation as throwing light upon the *normal* cholesterol function of the gall-bladder.

Some carefully controlled experiments upon the function of the gall-bladder with regard to bilirubin and cholesterol were made by Riegel, Johnston, and Ravdin (loc. cit.), utilizing dogs. A technique was employed which largely eliminated trauma to the gall-bladder; furthermore, since the experiments were quantitative, they avoided inaccuracies arising out of the use of the cholesterol-bilirubin ratio. A fine soft rubber catheter was passed up the common and cystic ducts into the gall-bladder, and the end of the catheter was brought to the exterior. A ligature was then applied around the central hepatic duct distal to the cystic duct. Damage to the cystic blood- and lymph-vessels was thus avoided, while the greater part of the bile continued to find its way into the duodenum. Their experiments dealt with an organ physiologically normal as judged by its ability to transform the hepatic bile of another dog into a fluid having the same composition as the bile which it had itself contained at the beginning of the experiment. Hepatic bile

was obtained from another animal, its cholesterol and pigment content determined, and a definite quantity introduced through the catheter into the gall-bladder. After a few hours it was withdrawn and changes in its volume and composition were determined. From these studies it was concluded that the normal gall-bladder does not resorb pigment, though this disappears rapidly if inflammation be present. In all of the uncomplicated experiments there was a slight decrease (10 per cent) in the cholesterol content of the gall-bladder, but where infection supervened the amount of cholesterol increased 96 per cent in twenty-four hours. Similar gross increases occurred when accessory hepatic ducts were present. The results clearly demonstrate that unless the occurrence of inflammation and the presence of accessory bile-ducts can be unequivocally excluded, no experimental evidence that the gall-bladder secretes cholesterol is admissible. The authors interpret their findings conservatively and incline to regard the 10 per cent diminution in the cholesterol content as within the limits of experimental error. The results, however, with regard to the cholesterol function of the gall-bladder are certainly more compatible with a resorptive than with a secretory process.

In conclusion, the balance of evidence obtained from the extensive series of experiments described indicates that the normal mammalian gall-bladder resorbs cholesterol from the bile. If, however, the gall-bladder be inflamed or traumatized, then effusion of blood-serum will greatly increase the amount of cholesterol in its contents.

b. Evidence Based upon Estimations of Biliary Cholesterol in Human Cases.—In a series of operations upon human cases of gall-bladder disease Elman and Graham took the opportunity of comparing the bile in the excised gall-bladder with that aspirated from the common hepatic duct during operation. The cholesterol-bilirubin ratio was determined in the two specimens of bile, and was found to be higher in the sample obtained from the gall-bladder. This was held to support their thesis that the gall-bladder secretes cholesterol, but it is clear that, before these estimations could be accepted as evidence, many fallacies would require to be excluded.

Chiray and Hesse (1932) analysed specimens of human bile obtained by duodenal rubage and found that the proportion of cholesterol to pigment was greater in the *B* specimen, derived supposedly from the gall-bladder, than in the *A* or *C* fractions which have their reputed source directly in the liver.

Accordingly, the small amount of evidence from human cases indicates that during the sojourn of bile in the gall-bladder, the concentration of cholesterol increases in greater measure than the concentration of bile-pigment. This, of course, falls far short of proof that the gall-bladder secretes cholesterol.

c. Indirect Evidence from Experiments on Resorption of Substances other than Cholesterol.—There has been much experimental work devoted to study of the ability of the gall-bladder to resorb a great variety of substances other than cholesterol and bile-pigments; these, being most closely germane to the question of cholesterosis, have been considered first. We cannot, of course, say that since the gall-bladder is able to resorb sodium chloride, therefore it also must resorb cholesterol, but as we shall see, the results of this great body of experimental work are more in keeping with resorption than with secretion of the latter. The difficulty of precise demonstration of the resorption of cholesterol contrasts sharply with the finality of experiments dealing with many other widely differing substances.

The water-resorbing function of the gall-bladder has been well recognized since the experiments of Rous and McMaster, and generally regarded as remarkable, though it is possibly not after all very extraordinary when it is compared with the daily performance of the analogous intestinal epithelium or the herculean labour of the renal epithelium. Demmel and Brummelkamp (1924) proved that the gall-bladder of the dog resorbs neutral fat, and Mentzer (1925) demonstrated fat in the epithelium of the gall-bladder of the rabbit twenty-five minutes after its introduction into the lumen. Whitaker (1929), using iodized oil, demonstrated it histologically in the epithelium of the gall-bladder of the cat and in the subjacent lymphatics, and showed that it may disappear in the course of a few weeks. Rosenthal and Licht (1928) demonstrated that the gall-bladder resorbs bile-acids, the disappearance of which could be accelerated by production of aseptic chemical inflammation. Iwanaga (1923), Mentzer (1925), and Nakashima (1926) showed that various types of dyes are rapidly resorbed. Nakashima found that the rate of resorption depends upon the diffusibility of the particular dye used rather than upon chemical composition, and Iwanaga demonstrated once again that the resorptive processes are accelerated by inflammation. Sweet (1929) found that sodium iodide in solution may disappear from the lumen of the gall-bladder within an hour, and Winkenwerder (1930) proved that ferrous ammonium citrate is very rapidly resorbed by the uninjured epithelium of the gall-bladder. Johnston (1931) showed that if sodium tetraiodophenolphthalein be introduced into the gall-bladder of a dog, 50 per cent may be resorbed in twenty-four hours. Andrews and Hrdina (1931) confirmed the findings of Mirovisch, Sachs, and Schrire (1930) that the gall-bladder has the power of resorbing calcium, and the latter group of investigators regarded this as so active a process that it might constitute a sort of internal cycle of calcium metabolism.

Secretion by the Human Gall-bladder.—When the gall-bladder is drained externally, the amount of fluid obtained depends upon the patency or otherwise of the cystic duct. If the latter be obstructed the daily drainage consists merely of a few c.c. of mucoid fluid. That the gall-bladder secretes mucus is well known. Phemister (1931) has shown that calcium carbonate passes into the human gall-bladder during periods when the cystic duct is obstructed by a stone, and may ultimately form a stucco-like mass filling the lumen. However, in gall-bladders containing this calciferous fluid (Kalk-milch-galle) the mucosa is usually the seat of advanced degenerative changes, and it may be difficult even to identify an epithelial layer, so that these observations do not establish the secretion even of calcium as one of the *normal* functions of the gall-bladder. Kerr and Lendrum (1936) have published an extraordinarily interesting case in which the presence in a gall-bladder of a papilloma composed of heterotopic intestinal epithelium was associated with profuse secretion of a fluid rich in sodium chloride. Such secretion has never been observed, however, in the normal gall-bladder.

The results of these extensive experiments and observations upon the physiology of the normal gall-bladder serve to establish beyond doubt that the activity of the latter with regard to a great variety of substances is resorption, and that the resorption is often accelerated by the hyperæmia and increased permeability of inflammation. If argument by analogy be permissible, it is reasonable to deduce that the normal function of the gall-bladder with regard to cholesterol will be resorptive also. This point will be discussed more fully later, from the histological aspect.

There is, it may be mentioned, a school of opinion represented by Sweet (1924), Blond (1927), and Halpert (1929), who have maintained that the normal gall-bladder does not merely concentrate the bile by taking up water, but actually resorbs all that enters during the inter-prandial period by the cystic duct, which functions as a one-way channel, and that it is at least unusual for any bile to be discharged from the gall-bladder down the cystic duct. Having once entered the gall-bladder the biliary constituents, including of course cholesterol, reach the intestine only after resorption, and re-excretion by the liver. Acceptance of this extreme view would clearly entail revision of all the accepted beliefs regarding the function of the gall-bladder and the mechanism of the expulsion of gall-stones from the gall-bladder and their passage along the ducts. None the less the ample evidence submitted of the very catholic resorptive capacity of the gall-bladder makes it appear at least possible that under certain circumstances the contained bile may be totally resorbed.

d. Evidence from Blood Cholesterol Studies in Human Cases and in Animals.—In the light of the accumulating evidence that only a small proportion of the cholesterol which daily passes out in the stool comes from the bile (Sperry, 1926; Sperry and Angevine, 1932), it is hardly to be expected that studies of cholesterol in the blood-plasma before and after cholecystectomy will show changes sufficiently marked to be of value in determining the role of the gall-bladder with regard to biliary cholesterol. In point of fact, the evidence is conflicting. Boyd (1923), using rabbits on a cholesterol-rich diet, found that following cholecystectomy the blood-cholesterol fell. Sweet (*loc. cit.*), on the other hand, found higher values in dogs after this procedure. Fowweather and Collinson (1926), studying the relation of hypercholesterolaemia to cholelithiasis in the human subject, found the average value of the plasma cholesterol in 11 cases to fall following cholecystectomy from 205 to 154 mg. per 100 c.c. If at all significant, the findings of Boyd and of Fowweather and Collinson would indicate that the gall-bladder resorbs cholesterol, those of Sweet that it secretes.

e. The Cholesterol Function of the Gall-bladder in the Light of Histological Observations.—The mucosa of the gall-bladder is lined by an epithelium of very uniform structure. It consists of a single layer of tall columnar cells, eosinophilic in reaction, with basally situated oval nuclei. Here and there a few goblet cells are scattered, relieving the simple monotony of the lining; in chronically inflamed gall-bladders they are much increased in number. The function of these goblet cells is beyond dispute—they are the source of a part of the mucus which is added to the bile during its sojourn in the gall-bladder, the remainder coming from well-formed muciniferous glands near the neck. The goblet cells therefore may be excluded from further consideration.

With regard to the function of the 'principal cells' of the mucosa, it may be observed that in the human body secretory and resorbing epithelia are sharply differentiated, and it is difficult to discover any instance of single epithelial cells that both secrete and resorb, although, e.g., in the intestine, cells of different types, some secretory, some resorptive, may be associated. In the gall-bladder lining, however, the outstanding histological feature is the structural homogeneity of the principal cells, and it is reasonable to assume that this connotes a corresponding uniformity of function representing in summation the normal physiological function of the gall-bladder lining. Now, as has been shown above, it is established that

under both physiological and experimental conditions this function is active resorption. The only known 'secretion', apart from mucus, is calcium carbonate, which may accumulate in pathological gall-bladders when the cystic duct is obstructed. On the other hand, the *normal* gall-bladder is capable, under experimental conditions, of taking up large amounts of water, sodium chloride, calcium chloride, bile-salts, bile-pigments, fat, and a variety of 'foreign' chemical substances. It is therefore *prima facie* unlikely that these cells, whose resorptive capacity is so generous, should possess in addition the power to secrete cholesterol.

In cholesterosis, abnormal amounts of lipid may be found in the epithelial lining of the gall-bladder (*Fig. 394*), but characteristically by far the greatest



FIG. 394.—Cholesterosis of the gall-bladder. Paraffin section, stained haematoxylin and eosin. There is a rich deposit of lipid material in the columnar epithelium of the gall-bladder, the cells of which in places are bloated and 'foamy'. In this specimen there is no stromal infiltration, although invaginated epithelial acini simulate nests of stromal macrophages. ($\times 100$.)

amount occurs in collections of stromal macrophages (*Figs. 395-400*). These macrophages, elements of the reticulo-endothelial system, are scavenger cells, whose function is to segregate foreign substances, or normal metabolites present in excess of immediate requirements. In many pathological conditions they take up large amounts of lipid, and if sufficiently numerous they form yellow masses, which may be either generalized or local, according as the lipid excess is a widespread alteration in the composition of the body fluids, or represents merely a local accident. Generalized lipid infiltration is exemplified by the lipid histiocytoses, which are relatively rare—Gaucher's disease, Pick's disease, Hand-Schüller-Christian disease—and by xanthelasma and xanthoma diabetorum, in all of which the basic lesion is an upset of lipid metabolism and relative hypercholesterolaemia. Examples of localized deposit are provided by certain tumours, especially those of tendon-sheaths, and the osteoclastomata, by degenerating haematomata, cerebral softenings, certain cases of chronic pyosalpinx, and the

condition of fat necrosis of the breast. Cholesterosis comes definitely into the category of local lipid infiltration, and accordingly the source of the lipid excess



FIG. 395.—Frozen section of gall-bladder illustrated in Fig. 402, stained hæmatoxylin and scharlach R. The lipid material occurs as darkly staining masses in the stroma of the plicæ. ($\times 75$.)



FIG. 396.—The same section as illustrated in Fig. 395, photographed with a polarizing microscope. The doubly refracting lipid is brilliantly shown and its fluid crystalline character suggested. In order to show the outline of the section the axis of the analyser has been set slightly off complete obscuration. ($\times 75$.)

must be sought locally—and where if not in the bile? Now Illingworth has shown that in cholesterosis the bile in the gall-bladder may contain 967.5 mg. of cholesterol



FIG. 397.—Colour drawing of the frozen section of gall-bladder illustrated in Fig. 395. The lipid material, stained orange, is contained in varying amounts in macrophages in the stroma of the plicae. The stroma is cellular and appears oedematous, but there is no true inflammatory reaction. Red blood-corpuscles are stained green. ($\times 125$.)



FIG. 398.—Cholesterosis of the gall-bladder. Paraffin section, stained hematoxylin and eosin. The histology is poor owing to delay in fixation. The specimen shows in the pedunculated bulbous tip a collection of macrophages solidly infiltrated with lipids. There is no trace of inflammatory infiltration, or of thickening of the wall of the gall-bladder. ($\times 100$.)



FIG. 399.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. The mucous membrane is distorted by gross distension of the plicæ with masses of foam cells. This, however, is the sole departure from normal. There is no fibrosis no inflammatory infiltration, and no degeneration. ($\times 125$.)



FIG. 400.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. This gall-bladder contained stones. The wall is thickened, the muscularis is hypertrophied, and the mucosa heavily infiltrated with round cells. In the midst of the inflammatory tissue a solitary foam cell is seen standing out as a clear spot. ($\times 100$.)

per 100 c.c., and if more evidence were desired that the bile in cholesterosis is unusually rich in cholesterol it would be furnished by the fact that in more than a third of the cases of well-marked cholesterosis, gall-stones of cholesterol-rich type are present. The gall-bladder resorbs as much as it can of the supersaturated cholesterol, yet it is unable to prevent crystallization.

Furthermore, the fact that the greater part of the lipid deposit is in the stroma is hardly compatible with a secretory process. We do not find the secretion of a gland segregated in its stroma but in the cells which are producing the secretion, and in these cells the greatest amount and the largest globules occur near the free border. This is the direct reverse of the state of affairs in cholesterosis, which presumably therefore depends upon resorption.

2. EXPERIMENTAL PRODUCTION OF CHOLESTEROSIS

The ease of production of cholesterosis of the gall-bladder varies with the dietetic habits of the animal. Among herbivora, administration of gross excess of cholesterol leads to massive deposits in the liver, spleen, adrenals, aorta, heart, etc., and it is relatively difficult to produce lipid infiltration of the mucosa of the gall-bladder. In the case of carnivora, on the other hand, it appears to be comparatively easy to produce a condition similar to human cholesterosis.

Dewey (1916) administered cholesterol to rabbits by intraperitoneal injection. Massive deposits were formed, especially in the liver and in the heart, and around some of these necrosis was noted, but in only a few cases was there the slightest evidence of lipid infiltration of the mucosa of the gall-bladder. Blaisdell and Chandler (*loc. cit.*) used 15 rabbits and 10 dogs. For a prolonged period each animal received daily in its food 1 g. cholesterol suspended in egg-yolk. All the dogs showed cholesterosis of the gall-bladder, but only 4 of the rabbits developed this condition. Since cholesterosis did not occur in cases in which the cystic duct had been ligated, the investigators concluded that the mechanism underlying cholesterosis is resorption of lipid from the bile. Illingworth (1929) induced in rabbits a very high degree of cholesterolaemia (as much as 1136 mg. per 100 c.c.) by cholesterol feeding. In 2 of the animals chronic cholecystitis was produced by injection into the subserous coat of streptococci of low virulence. In these cholesterosis occurred. In 4 controls receiving dietetic treatment alone there was no lipid deposit in the gall-bladder. Patey (1934) performed similar experiments on 19 rabbits; in all massive lipid infiltration took place in aorta, liver, and other organs. Where laparotomy had been performed there was gross lipid infiltration of the healed abdominal wound. The gall-bladder, however, was spared unless it had been traumatized or infected, and the lipid deposit occurred only in the scar tissue or at the site of inflammatory change. Thus the deposit appeared more usually in the subserous coat than in the mucosa. It occurred whether or not the cystic duct was ligated. The lesions, however, did not bear a very close resemblance to human cholesterosis. Patey concluded that, in his experiments at all events, the lipid deposit was a xanthomatous condition dependent upon deposition from the blood, and that his results furnished no evidence regarding the debatable cholesterol function of the gall-bladder.

In the course of the work of Rousselot and Bauman (*loc. cit.*) upon the cholesterol function of the canine gall-bladder, a marked degree of cholesterosis occurred

following the introduction into the lumen for 48 hours in all of a 0.2 per cent solution of cholesterol in bile-salts. Increase in the cholesterol esters in the wall was accompanied by progressive and parallel disappearance of cholesterol from the contents of the gall-bladder.

3. IS CHOLESTEROSIS A SPECIFIC TYPE OF CHOLECYSTITIS ?

During the past few years the author has studied histologically about 400 gall-bladders removed by various surgeons ; among these 81 examples of cholesterosis were encountered, and in the majority the mucosal lipid deposit was sufficiently abundant to be seen with the unaided eye. The figures probably exaggerate the incidence of gross cholesterosis, for many of the cases were discovered during a study limited to the less severe grades of cholecystitis ; and cases of cholelithiasis were excluded from this series unless accompanied by cholesterosis (Mackey, 1934).

The general results of the histological examinations were, briefly, as follows : Lipoid infiltration occurred in the lining epithelium (*see Fig. 394*) or in the connective tissue of the mucosa, but more frequently and more abundantly in the latter (*see Fig. 398*). It was found in all types of gall-bladders ; in some there was no evidence of inflammatory change ; many were slightly thickened, and showed abundant infiltration with lymphocytes ; while a few, especially those in which stones were present, were so greatly thickened and fibrosed that the lipoid infiltration constituted a very minor feature of the histological picture. In one extreme example of the last type the mucosa was crowded with densely aggregated lymphoid follicles and the lipoid deposit was confined to a few isolated macrophages within these follicles (*see Fig. 400*). The degree of cholesterosis was never proportional to the degree of inflammatory change, and was rare if the latter was severe. In short, the histological picture presented by cholesterosis is so pleomorphic that it is hard to believe that it is a pathological entity. Moreover, since accompanying inflammatory changes vary so greatly in degree, and may indeed be completely absent, the etiological basis of cholesterosis is surely not inflammatory.

The work of Cappell (1930) and others has shown that cells of macrophage type arise probably by differentiation of lymphocytes. These occur abundantly throughout the alimentary tract under normal conditions, and their presence in moderate numbers in the wall of the gall-bladder is probably not pathological. Similarly the foam cells should be regarded not as evidence of an inflammatory process, but simply as visible evidence of a process of lipoid resorption, common both to normal gall-bladders and to those showing moderate chronic inflammatory changes. It is quite likely, moreover, that cholesterosis may in some cases be transitory, disappearing should the biliary cholesterol fall to a lower level, or the gall-bladder wall lose its capacity to resorb and store the lipoid.

The cholesterol polypi which are so common in cholesterosis of the gall-bladder may be formed to accommodate a local superabundance of lipoid, or they may represent secondary cholesterosis of pre-existing polypi. Both mechanisms probably operate. In support of the second, it may be mentioned that polypi without lipoid are occasionally seen (*Fig. 401*), but they appear to be peculiarly susceptible to cholesterosis, for much more often the polypi contain lipoid when the remainder of the gall-bladder shows none.

Before concluding this discussion of the histological evidence bearing upon cholesterosis and the cholesterol function of the gall-bladder, it must be mentioned once more that Elman and Graham believe that the gall-bladder secretes cholesterol, and that this process becomes more active in the presence of inflammation. This, however, is unlikely, for in the ordinary course inflammation of a secretory epithelium leads to diminution or cessation of its secretion, not to intensification, and still less to the development of a new and highly specialized secretion. Nor

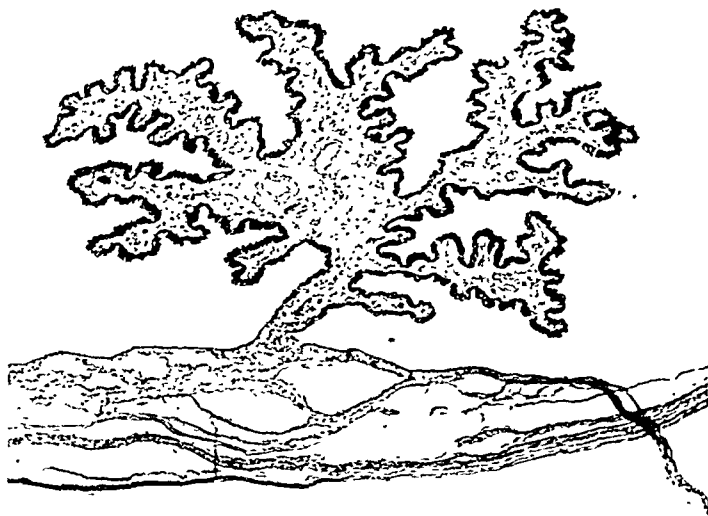


FIG. 401.—A polyp or papilloma occurring in a thin-walled degenerate hydropic gall-bladder. The polyp is covered with intensely staining short columnar epithelium. The stroma is well formed and vascular. There is no cholesterosis. ($\times 20$.)

can it be admitted, for reasons given above, that cholesterosis is the direct result of an inflammatory process in the wall of the gall-bladder, and, as will be discussed in a later section, it is unusual to find organisms in the strawberry gall-bladder.

To sum up, cholesterosis is not a manifestation of cholecystitis, or of cholesterol secretion by the gall-bladder, but of active resorption of cholesterol, from bile unusually rich in that substance, by a relatively normal mucous membrane.

4. GALL-STONE FORMATION IN CHOLESTEROSIS

In a recent study of certain aspects of gall-bladder disease, 67 cases of cholesterosis were encountered, and of these 21 bore stones (Mackey, loc. cit.). There were 8 cholesterol solitaires, and 7 sets of mulberry stones. In 4 cases, cholesterol-rich stones had superimposed mixed laminæ, presumably of inflammatory origin; in only one case did the stones appear to be *primarily* of 'mixed' type. These figures correspond with the general experience. In about a third of all cases, cholesterosis is complicated by the presence of gall-stones. Almost without exception these are of cholesterol-rich types, cholesterol solitaires and mulberry stones occurring with practically equal frequency. Although the cholesterol solitaire is equally

common, the mulberry stone has been regarded by many writers as characteristic of cholesterosis, especially since Lichtwitz's original suggestion that its genesis may be traced to the dehiscence of cholesterol polypi. Without doubt these polyps, in gross form, are not unlike pedunculated mulberry stones, but the heart of a mulberry stone is composed of pigment, while the cholesterol polyp is a collection of macrophages containing cholesterol esters in foamy emulsion. A transformation of the one into the other is hardly conceivable. I have seen many hundreds of tiny mulberry stones in a gall-bladder bearing only a few small polyps, where it seemed scarcely possible that the latter could have given rise to so multitudinous a progeny.

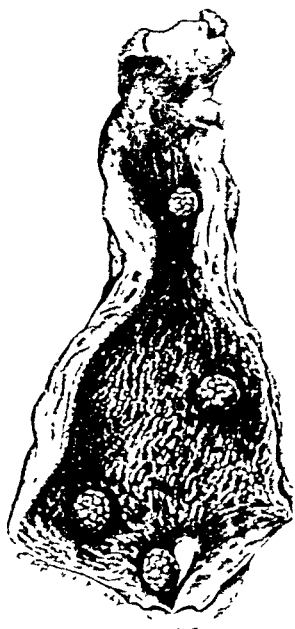


FIG. 402.—Cholesterosis of the gall-bladder with pigment and mulberry calculi. The gall-bladder is normal in size and thickness. The mucous membrane shows heavy infiltration of the longitudinal plicae with lipid material. In the lumen there are four typical mulberry stones and a number of tiny pigment concretions.



FIG. 403.—External aspect of specimen shown in Fig. 402. There is abundant fat in the subserous connective tissue. The hyperemia and the punctate hemorrhages are results of operative trauma.

Moreover, when mulberry stones are numerous it is often possible to separate them into a number of groups, in each of which the individuals are uniform in size and presumably therefore of equal age. A theory implying that in these circumstances polypi have been shed in crops separated by fairly long intervals is, to say the least, highly speculative. The specimen illustrated in Fig. 402 is an unusually valuable piece of evidence upon the question of the origin of the mulberry gall-stone. It contained 4 typical mulberry stones with pigment centres and a superficial lamina of cholesterol, and in addition a crop of tiny spicular pigment stones, some of which if retained in such a gall-bladder would surely form the nuclei of the succeeding brood of mulberry stones. The Lichtwitz theory is attractive but untenable, and, on the whole, the smooth radially arranged mulberry stone with its soft pigment

centre is best explained as the end-result of a special type of cholesterol crystallization, upon an irregular pigment nucleus. Illingworth (1936) has recently expressed a similar opinion.

Rovsing (1925), indeed, traces the origin of all gall-stones, even the cholesterol *solitaire*, to crystallization of cholesterol upon nuclei of 'bilihumin', one of the less soluble bile-pigments. This he insists is not a constituent of normal bile, but is produced in the liver in diseased and toxic states, and frequently during pregnancy. In the bile capillaries it forms irregular concretions, which are swept down by the

bile, often presumably in showers, to the larger ducts and the gall-bladder. It is certain that the vast majority of all gall-stones have a kernel of pigment—even on occasion the cholesterol *solitaire*. Some *solitaires* do not have a pigment nucleus, and Rovsing suggests that the pigment originally present has disappeared by diffusion. It may well be, however, that the solitary cholesterol stone may arise as a result of simple crystallization from bile containing a gross excess of cholesterol. If at a time when the gall-bladder bile becomes supersaturated with cholesterol, the gall-bladder happens to contain a few pigment concretions, the cholesterol will naturally separate out on their surfaces, and give rise to mulberry calculi. New broods of stones are laid down each time the arrival of pigment nuclei in the gall-bladder coincides with the occurrence of an excess of cholesterol in the bile. When the stones become numerous and occupy the greater part of the lumen of the gall-bladder, they become faceted (cf. Fig. 404).

In view of the frequency with which we find the cholesterol *solitaire* in the strawberry gall-bladder it is quite clear that there is no specific connection between cholesterosis and mulberry gall-stones. The latter are merely cholesterol-rich stones whose character has been determined by the shape of their pigment nuclei, by the number of these nuclei in relation to the size of the gall-bladder, and by the complex variations in the character of the mother-liquor. Cholesterosis might equally well occur in association with multiple faceted stones were it not that, generally speaking, the latter, by their very numbers, have set up pathological changes in the wall of the

gall-bladder so severe that it has lost the capacity to develop cholesterosis.

It is generally accepted that the '*solitaire*' calculi, so frequent in cholesterosis, arise aseptically in non-inflamed gall-bladders by crystallization from bile supersaturated with cholesterol. This is in itself an argument against the existence of an inflammatory basis for cholesterosis.

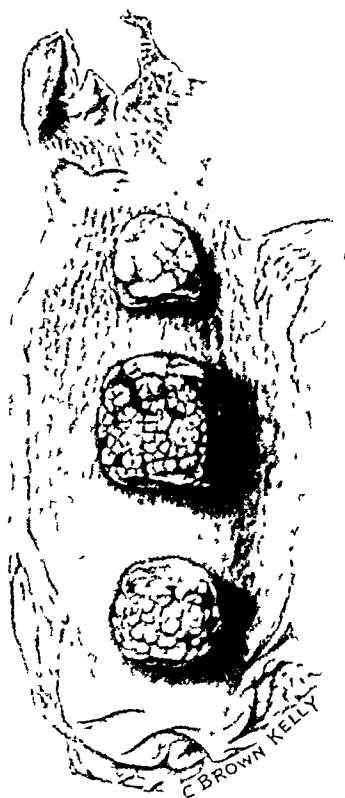


FIG 404.—Cholesterosis of the gall-bladder with three large cholesterol-rich stones. The lipid deposit is moderate in amount, and it has been rendered less prominent by lytic changes following removal of the gall-bladder. The three stones are of considerable age. They illustrate how, when stones come to occupy the greater part of the cavity of the gall-bladder, facets form on contiguous surfaces.

To conclude, cholesterosis and lithiasis are cognate manifestations of supersaturation of the bile with cholesterol, not cause and effect. Cholesterosis, however, is not without important significance, for it certainly betokens a metabolic state with an unduly high level of biliary cholesterol, and likely therefore to lead to gall-stone formation. The etiology of the characteristic stones, both 'solitaire' and 'mulberry', is primarily metabolic, not infective, although inflammatory complications of mechanical origin are likely to supervene later.

5. THE BACTERIOLOGY OF THE GALL-BLADDER IN CHOLESTEROSIS

On this point published figures are few. Illingworth reported 35 cases, 17 with stones. Of this total 29 proved sterile, 3 harboured streptococci of avirulent types, and 2 coliform bacilli. In the course of a recent bacteriological investigation by the author of 100 unselected gall-bladders removed at operation by various surgeons, the gall-bladders were dissected under aseptic precautions, and cultures, aerobic and anaerobic, on solid and fluid media, were made from the various layers of the wall, from the cystic lymph-node when this was present, and from the bile. In the 14 cases of cholesterosis encountered in this series the absence of infection was very striking, for in 13 no growth was obtained from any of the materials examined, and in the remaining 1 a non-hæmolytic streptococcus was found. It may in fact be said that in simple cholesterosis the bacterial flora of the gall-bladder is not more rich than that of gall-bladders which, on histological grounds, would be regarded as practically normal.

6. CHOLECYSTOGRAPHY IN CHOLESTEROSIS

According to Rossi (1932), who, incidentally, believes that cholesterosis is a pathological manifestation and is accompanied by symptoms, the cholesterol gall-bladder may be visible in the direct film of the upper abdomen. He states further that following administration of the cholecystographic dye the shadow appears slowly, is ill-defined, and may be mottled; after a fatty meal it disappears quickly, often with an exacerbation of pain. So far as the author knows this association of pain with cholesterosis has not been confirmed. Illingworth (1929) found that in the absence of other evidence of definite cholecystitis, the cholesterosis gall-bladder produces a dense normal shadow, and that contraction is active following the fatty meal. The author's observations previously reported (*loc. cit.*) may be reproduced here in tabular form (*Table I*). They concern 58 cases of cholesterosis, 20 complicated by the presence of stone.

Table I.—CHOLECYSTOGRAPHIC RESPONSE IN CHOLESTEROSIS

TYPE OF GALL-BLADDER LESION	TOTAL CASES	NORMAL DENSITY	FAINT	NO SHADOW
Cholesterosis	38	9	25	4
Cholesterosis with stone(s) ..	20	0	17	3

These figures demonstrate that, in the absence of stones and of secondary cholecystitis, the gall-bladder showing cholesterosis is frequently capable of achieving a normal concentration of bile and dye. The rapid diminution in size after the fat meal mentioned by Rossi and Illingworth indicates either brisk contraction of the gall-bladder musculature, or, as Halpert, Sweet, and Blond would have it, rapid resorption of the contents of the gall-bladder for re-excretion by the liver. In any case it betokens a gall-bladder functionally not far removed from normal.

7. CLINICAL CONSIDERATIONS

It has never been possible to associate with cholesterosis a pathognomonic symptom-complex; the complaints attributed to it have always been those generically termed 'cholecystitic'. In some cases there has been a history of biliary colic, in others of 'gall-bladder dyspepsia'. Such symptoms, however, occur in all types of derangement of the biliary apparatus, both functional and organic, and probably some of them may occur also in upsets of other parts of the alimentary tract. Reports of clinical results following cholecystectomy have been conflicting, and this is not surprising, since, as has been shown, cholesterosis is a histological feature that may be found in gall-bladders otherwise normal, or in association with lesions in all degrees of severity.

Moynihan's paper, "A Disease of the Gall-bladder Requiring Cholecystectomy", has exerted a considerable influence upon subsequent surgical practice. He had been accustomed to perform simple cholecystostomy in cases presenting symptoms referable to the gall-bladder but in which at operation no gall-stones were found. Six cases were recorded in which this treatment produced no benefit, and which were subsequently relieved of their pain by cholecystectomy. In each the mucosa of the gall-bladder was marked by yellow stippling, and the coincidence seemed so striking that Moynihan attributed the pre-operative symptoms to this abnormality. It has been shown, however, that true biliary pain will be cured in a large proportion of cases by cholecystectomy, even if the gall-bladder be practically normal; it is not correct, therefore, to assume that the symptoms in these cases were the result of the presence of cholesterosis, and still less that cholesterosis invariably produces painful symptoms. The correct interpretation is simply that biliary pain, unrelieved by cholecystostomy, may in some cases be cured by cholecystectomy. Judd and Mentzer (1927), in a study of 1000 cases of cholesterosis treated by cholecystectomy, found that good results were achieved only if pain had been a dominant symptom. White and Riddick (1928) concluded that cholesterosis is only part of a general metabolic dysfunction, and that cholecystectomy is therefore unlikely to lead to relief. Stanton (1932) had 6 cases of cholesterosis in his series of 93 stoneless cases, and of these 4 were not benefited by cholecystectomy. On the other hand, Young (1928) had 45 strawberry gall-bladders in his large series, and formed the impression that "some of the worst cases, with a marked tendency to myocardial degeneration, were strawberry gall-bladders, and some of the most marked relief was achieved after removal of these". Such occurrences are among the happy mysteries of biliary surgery. From time to time one encounters reports describing relief of rheumatoid arthritis, myocarditis, or nephritis following removal or drainage of a gall-bladder which showed no very gross lesion, or possibly contained a cholesterol solitaire. Kopp (1929) alleviated

by cholecystectomy the symptoms of 7 out of 11 cases of cholesterosis without stone. Gosset (1928) reported improvement in 13 of 16. Illingworth (1929) could trace only 6 of his 35 cases, and these over a relatively short period, but all appeared to be well.

In a recent investigation, already quoted, of the end-results after cholecystectomy in minor degrees of gall-bladder disease, the post-operative histories were traced of 52 cases of cholesterosis, 19 with and 33 without gall-stones (Mackey, loc. cit.). These are summarized in *Table II*.

Table II.—THE CLINICAL RESULT AFTER CHOLECYSTECTOMY IN CASES OF CHOLESTEROSIS

TYPE OF LESION	TOTAL	WELL	IMPROVED	UNIMPROVED	POST-OPERATIVE DEATH
Cholesterosis without stone ..	33	14	2	16	1
Cholesterosis with stone(s) ..	19	6	13	0	0

All the cases with gall-stones were at least improved after removal of the gall-bladder, but of the stoneless cases half were unbenefited. The results in this latter category were no more satisfactory than those in a larger series in which the histological changes in the gall-bladder were minimal. The end-results of cholecystectomy for gall-stones will always be better than those in 'cholecystitis without stone', for in a proportion of the latter the pre-operative complaints have not had their origin in the gall-bladder. In this connection it should be emphasized that cholesterosis *per se* probably does not produce symptoms, and certainly none likely to be relieved by cholecystectomy. Further, the end-results shown above were closely related to the character of the pre-operative complaints. Patients who had suffered before operation from true gall-bladder colic were almost always improved, but in those with more vague complaints the outcome was often unsatisfactory. It is probable that true biliary colic may occur, even apart from organic disease, in functional derangements of the gall-bladder (Westphal, 1923), and that in most of these cases it will be relieved by cholecystectomy. On the other hand, it is quite likely, in cases with vague dyspeptic symptoms, that the latter have had their origin outside the gall-bladder. Consequently, little benefit is to be expected from cholecystectomy in such indefinite cases. Subjective symptoms are of course by no means proportional to the severity of gall-bladder lesions, and depend greatly upon the psychological constitution of the patient.

In view of the frequency with which gall-stones complicate cholesterosis the suggestion may be made that when the gall-bladder is the seat of this lesion it should be removed, in order to *prevent* gall-stone formation. This is surely an undesirably radical and dangerous form of treatment for a condition that appears to be merely an unusual manifestation of a normal physiological process.

It is clear, however, that to discuss the surgical treatment of cholesterosis *per se* is really beside the point, for the diagnosis of cholesterosis cannot be made until the gall-bladder is exposed at operation and opened. If this operation has been dictated by typical gall-bladder symptoms, such as recurring biliary colic, or

if at operation gall-stones are found, then the correct treatment is cholecystectomy, whatever the condition of the gall-bladder mucosa. If, on the other hand, symptoms have been vague or atypical, and the operation is in a sense exploratory, then the mere presence of uncomplicated cholesterosis neither calls for, nor justifies, cholecystectomy, for there is no evidence that it is ever responsible for the production of symptoms.

SUMMARY AND CONCLUSIONS

A review is presented of the subject of cholesterosis of the gall-bladder, supplemented by personal observations of 87 cases.

'Cholesterosis' is the term applied to a common condition in which the mucosa of the gall-bladder is infiltrated with lipid substances. In its nature it is related to the xanthomata.

It is not a specific lesion, but rather a histological feature that may occur at random in gall-bladders showing all grades of pathological change; in fact, apart from lipid infiltration, many are normal. In the presence of advanced inflammatory or degenerative changes, cholesterosis is rare.

It is probable that the gall-bladder has the power of resorbing cholesterol from the bile. When the cholesterol is unusually abundant the process may become, as it were, visualized, and cholesterosis result. Should the amount of cholesterol in the bile diminish later, the cholesterosis probably may disappear.

The gall-bladder showing simple cholesterosis rarely harbours a demonstrable organismal infection.

In a third of the cases of cholesterosis, gall-stones occur; these are almost always of cholesterol-rich types. The cholesterosis and the stones are not respectively cause and effect, but are parallel results of super-saturation of the bile with cholesterol. Cholesterosis betokens a state of affairs in which gall-stones are liable to be formed, but it is not otherwise of clinical significance.

Cholesterosis does not, *per se*, produce symptoms. Consequently, the mere presence of lipid infiltration of the mucosa of the gall-bladder does not justify cholecystectomy, nor does it support a prediction that the clinical result of this operation will be gratifying.

Acknowledgements.—This paper is principally the result of work begun in 1933 at St. Louis, when the author was working under the guidance of Professor Evarts A. Graham. It was at his suggestion that the study of cholesterosis was undertaken, and to him warm thanks are due for clinical and pathological facilities unstintingly furnished, and not less for his sustained and stimulating interest in the investigation, an interest unimpaired by the fact that the author's views on the cholesterol function of the gall-bladder were, and are, at variance with his own.

Some of the early pathological material was obtained in the Pathological Institute of the Western Infirmary, Glasgow. The three specimens illustrated by Miss Brown Kelly's admirable colour drawings are in the Museum of the Department of Surgery, University of Glasgow. For the use of these and for his interest and encouragement I have to thank my chief, Professor Archibald Young.

The photomicrographs (Figs. 397, 398, 400, 401, 403) and the photograph (Fig. 392) are by Mr. John Kirkpatrick. This last is from an illustration in Sir Robert Muir's *Textbook of Pathology* (Edward Arnold).

I am indebted to Professor Browning for a great deal of valuable constructive criticism during the writing of the paper, and to Miss A. M. Kelly for her excellent secretarial work.

BIBLIOGRAPHY

- ANDREWS, E., and HRDINA, L., "Absorption of Calcium by the Gall-bladder", *Amer. Jour. Med. Sci.*, 1931, clxxxi, 478.
- ANDREWS, E., DOSTAL, L. E., and HRDINA, L., "Etiology of Gall-stones. Is Cholesterol Secreted by the Gall-bladder Mucosa?", *Arch. of Surg.*, 1933, xxvi, 382.
- ASCHOFF, L., "Zur Frage der Cholesterinbildung in der Gallenblase", *Munch. med. Woch.*, 1906, iii, 1847.
- ASCHOFF, L., and BACMEISTER, A., "Die Cholelithiasis", 1909, p. 14. Jena.
- BLAISDELL, F. E., and CHANDLER, L. R., "The Relation Between Cholesterolæmia and Deposits of Cholesterol in the Gall-bladder", *Amer. Jour. Med. Sci.*, 1927, clxxiv, 492.
- BLOND, K., "Zur Gallenblasenphysiologie und Pathologie", *Klin. Woch.*, 1927, vi, 1606.
- BOYD, W., "Gall-bladder Problems", *Canad. Med. Assoc. Jour.*, 1922, xii, 689.
- BOYD, W., "Studies in Gall-bladder Pathology", *Brit. Jour. Surg.*, 1923, x, 337.
- BRAND, J., "Beitrag zur Kenntnis der menschlichen Galle", *Arch. f. d. g. Physiol.*, 1902, xc, 491.
- CAPPELL, D. F., "Intra-vitam and Supra-vital Staining", *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 429.
- CHIRAY, M., and HESSE, J., "The Role of the Gall-bladder in the Secretion of Cholesterol", *Presse méd.*, 1932, xl, 1445.
- CORKERY, J. R., "Chronic Catarrhal Cholecystitis with Lipoid Deposit", *Ann. of Surg.*, 1922, lxxvi, 736.
- DEMME, R., and BRUMMELKAMPF, K., "An Experimental Study of Gall-bladder Function", *Mittteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1924, xxxvii, 515.
- DEWEY, K., "Experimental Hypercholesterolæmia", *Arch. of Internal Med.*, 1916, xvii, 757.
- ELMAN, R., and GRAHAM, E. A., "The Pathogenesis of the Strawberry Gall-bladder", *Arch. of Surg.*, 1932, xxiv, 14.
- ELMAN, R., and TAUSSIG, J. B., "The Cholesterol Function of the Gall-bladder", *Jour. of Exper. Med.*, 1931, liv, 775.
- FOWWEATHER, F. S., and COLLINSON, G. A., "Gall-stones and Hypercholesterolæmia", *Brit. Jour. Surg.*, 1926, xiv, 583.
- GOSSET, A., BERTRAND, I., and LOEWY, G., "La Vésicule fraise", *Progrès méd.*, 1928, No. 44, p. 1792.
- HALPERT, B., "The Gall-bladder, its Functions and some of Their Disturbances in the Light of Recent Investigations", *Arch. of Surg.*, 1929, xix, 1037.
- ILLINGWORTH, C. F. W., "Cholesterosis of the Gall-bladder", *Brit. Jour. Surg.*, 1929-30, xvii, 203.
- ILLINGWORTH, C. F. W., "The Formation of Gall-stones", *Edin. Med. Jour.*, 1936, xliii, 481.
- IWANAGA, H., "Experimentelle Studien über das Resorptionsvermögen der Gallenblase", *Mittteil d. med. Fak. Univ. Kyushu, Fukuko*, 1923, vii, 1.
- JOHNSTON, C. G., "Absorption of Tetraiodophenolphthalein by the Gall-bladder", *Jour. Clin. Investigation*, 1931, x, 9.
- JUDD, E. S., and MENTZER, S. H., "Cholesterosis of the Gall-bladder", *Collected Papers of the Mayo Clinic*, 1927, xix, 310.
- KERR, A. B., and LENDRUM, A. C., "A Chloride-secreting Papilloma in the Gall-bladder", *Brit. Jour. Surg.*, 1936, xxxiii, 615.
- KOPP, J. G., "Die Erdbeergallenblase", *Arch. f. klin. Chir.*, 1928, cli, 411.
- KUSNETOWSKY, N., "Ueber die Fettresorption und Ausscheidung durch das Epithel der Gallengänge", *Mittteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1923, xxxvii, 145.
- LAROCHE, G., and FLANDIN, CH., "Recherche histologique de la Cholestérine dans la Bile et les Parois de la Vésicule biliaire", *Semaine méd.*, 227.
- LICHTWITZ, L., "Ueber die Bildung der Harn- und Gallensteine", *Ergebn. d. inn. Med. u. Kinderheilk.*, 1914, xiii, 1.
- LOEWY, G., "Vésicule et Cholestérol. Absorption ou Sécrétion?" *Arch. des Mal. de l'App. dig.*, 1932, xxii, 937.
- MACCARTY, W. C., "The Pathology of the Gall-bladder, and Some Associated Lesions", *Ann. of Surg.*, 1910, li, 651.

- MENTZER, S. H., "Cholesterosis of the Gall-bladder", *Collected Papers of the Mayo Clinic*, 1925, xvii, 149.
- MACKEY, W. A., "Cholecystitis Without Stone", *Brit. Jour. Surg.*, 1934, xxii, 274.
- MIROVISCH, G., SACHS, G., and SCHRIRE, T., "The Absorption of Calcium by the Gall-bladder", *Jour. of Physiol.*, 1930, lxx, 434.
- MOYNIHAN, B. G. A., "A Disease of the Gall-bladder Requiring Cholecystectomy", *Ann. of Surg.*, 1909, l, 1265.
- NAKASHIMA, K., "Absorption of Dyes in the Gall-bladder of the Rabbit", *Acta Schol. med. Univ. Imp. Kyoto*, 1926, ix, 225.
- PATEY, D. H., "The Experimental Production of Cholesterosis", *Brit. Jour. Surg.*, 1934, xxii, 378.
- PHEMISTER, D. B., REWBRIDGE, A. G., and RUDISHILL, H., jun., "Calcium Carbonate Gall-stones and Calcification of the Gall-bladder following Cystic Duct Obstruction", *Ann. of Surg.*, 1931, xciv, 493.
- POLICARD, A., "Recherches histochimiques sur les Substances grasses absorbées au Niveau de la Vésicule biliaire", *Comptes rend. Soc. de Biol.*, 1914, lxxvi, 518.
- RIEGEL, C., RAVDIN, I. S., and JOHNSTON, C. G., "Absorption from the Bile-free Gall-bladder", *Amer. Jour. Physiol.*, 1932, xciv, 638.
- ROSENTHAL, F., and LICHT, H., "Die Resorption der Gallensäuren in der normalen und entzündeten Gallenblase", *Klin. Woch.*, 1928, vii, ii, 1952.
- ROSSI, A., "Atonic Gall-bladder and Strawberry Gall-bladder", *Amer. Jour. Roentgenol.*, 1932, xxvii, 205.
- ROUS, P., and MCMASTER, P. D., "Physiological Causes for the Varied Character of Stasis Bile", *Jour. of Exper. Med.*, 1921, xxxiv, 75.
- ROUS, P., and MCMASTER, P. D., "The Concentrating Activity of the Gall-bladder", *Jour. of Exper. Med.*, 1921, xxxiv, 47.
- ROUSSELOT, L. M., and BAUMAN, L., "Experimental Production of Cholesterosis of the Gall-bladder", *Surg. Gynecol. and Obst.*, 1934, lxi, 585.
- ROVSING, P., *Calculs Biliaires*, 1925. Paris: Masson et Cie.
- SPERRY, W. M., "Lipid Excretion. IV. Study of the Relationship of the Bile to the Faecal Lipids, with Special Reference to Certain Problems of Sterol Metabolism", *Jour. of Biol. Chem.*, 1926-27, lxxi, 351.
- SPERRY, W. M., and ANGEVINE, "The Secretion of Lipids into the Intestine", *Jour. of Biol. Chem.*, 1932, 96, 769.
- STANTON, E. MACD., "The Stoneless Gall-bladder. A Study of Operative Cases", *Amer. Jour. Surg.*, 1932, xviii, N.S., 246.
- SWEET, J. E., "The Gall-bladder: Its Past, Present, and Future", *Internat. Clinics*, 1924, i, 187.
- SWEET, J. E., "The Physiology of the Gall-bladder", *Ann. of Surg.*, 1929, xc, 937.
- TORINOUMI, K., "Woher stammt das Cholesterin des Gallensteins?", *Beitr. z. pathol. Anat. u. allg. Pathol.*, 1923, lxxii, 456.
- VIRCHOW, R., "Ueber das Epithel der Gallenblase und über einen intermediären Stoffwechsel des Fettes", *Arch. f. pathol. Anat., Physiol., klin. Med.*, 1857, xi, 574.
- WESTPHAL, K., "Muskelfunktion, Nervensystem und Pathologie der Gallenwege", *Zeits. f. klin. Med.*, 1923, xcvi, 22.
- WHITE, C. S., and RIDDICK, A. L., "Persistence of Symptoms after Cholecystectomy", *Southern Med. Jour.*, 1928, xxi, 857.
- WHITAKER, L. R., "Problems in the Normal and Abnormal Physiology of the Gall-bladder", *Arch. of Surg.*, 1929, xviii, 1783.
- WILKIE, A. L., and DOUBILET, H., "Passage of Cholesterol through the Mucosa of the Gall-bladder", *Arch. of Surg.*, 1933, xxvi, 110.
- WINKENWERDER, W. L., "A Study of Resorption from the Biliary Tract", *Johns Hopkins Hosp. Bull.*, 1930, xlv, 5272.
- YOUNG, E. L., jun., "The End-results of Chronic Cholecystitis", *New Eng. Jour. Med.*, 1928, cxcviii, 729.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

PROFESSOR GREY TURNER AT THE BRITISH POST-GRADUATE MEDICAL SCHOOL

THE foundation of this institution, which is the first of its kind in this country, marks a notable epoch in the history of British medical education. The hospital which affords its clinical basis is a modern building which will contain 534 beds when certain additions are complete. It was originally built as a poor-law hospital, and had hardly been completed in 1914 when it was taken over temporarily by the medical department of the War Office, and then used as a special military orthopædic hospital, where Sir Robert Jones had the centre of those training and organizing activities which were of such inestimable benefit to our wounded in the War. It was transferred in 1929 to the London County Council, and represents the most recent of all the many hospitals controlled and administered by that body. It is situated in Ducane Road, Shepherd's Bush, Hammersmith, on the westerly fringe of greater London. The idea of a post-graduate medical school was first initiated by the Right Hon. Christopher Addison in 1921 when he was Minister of Health. After long and careful consideration of various possibilities it was decided to found the new school under the ægis of the London University in affiliation with the London County Council hospital at Hammersmith. This, too, marks a very significant development in the hospital policy of this country, viz., the assumption of the highest type of clinical research and training by a municipal hospital in association with a University. So that already this Hammersmith hospital has made history both during the war and now in the initiation of post-graduate teaching.

The medical school, whose charter was received in 1931, was formally opened by King George V on May 13, 1935. The teaching work is divided up into five departments: Medicine, Surgery, Obstetrics with Gynæcology, Pathology, and Radiology. The staff, apart from consultants called in occasionally, is divided into two categories: the one consists of whole-time professors, readers, and assistants, and the other of selected teachers from other medical schools who give lectures and clinical courses on special subjects from time to time. The permanent staff of the present surgical unit is as follows:—

Director : Professor G. Grey Turner.

Reader : Mr. A. K. Henry.

Senior Assistant (L.C.C.) : Mr. D. L. Lewis.

First Assistant (Orthopædics) : Mr. A. J. Watson.

First Assistant (Genito-urinary) : Mr. A. L. Light.

First Assistant (Genito-urinary) : Mr. G. Y. Feggetter.

First Assistant (Gastro-enterology) : Mr. R. H. Franklin.

Assistant (E.N.T.) : Mr. J. I. Griffiths.

For the current session the following teachers are giving special lectures and demonstrations :—

Mr. Geoffrey Jefferson : *Surgery of the Nervous System.*

Professor John Fraser : *Acute Bone Infections.*

Professor Seymour Barling : *Other Acute Surgical Infections.*

Mr. W. B. Gabriel : *Rectal Diseases.*

Professor Hey Groves : *Fractures.*

Mr. J. E. H. Roberts : *Surgery of the Chest.*

Mr. Russell Howard : *Diseases of the Breast.*

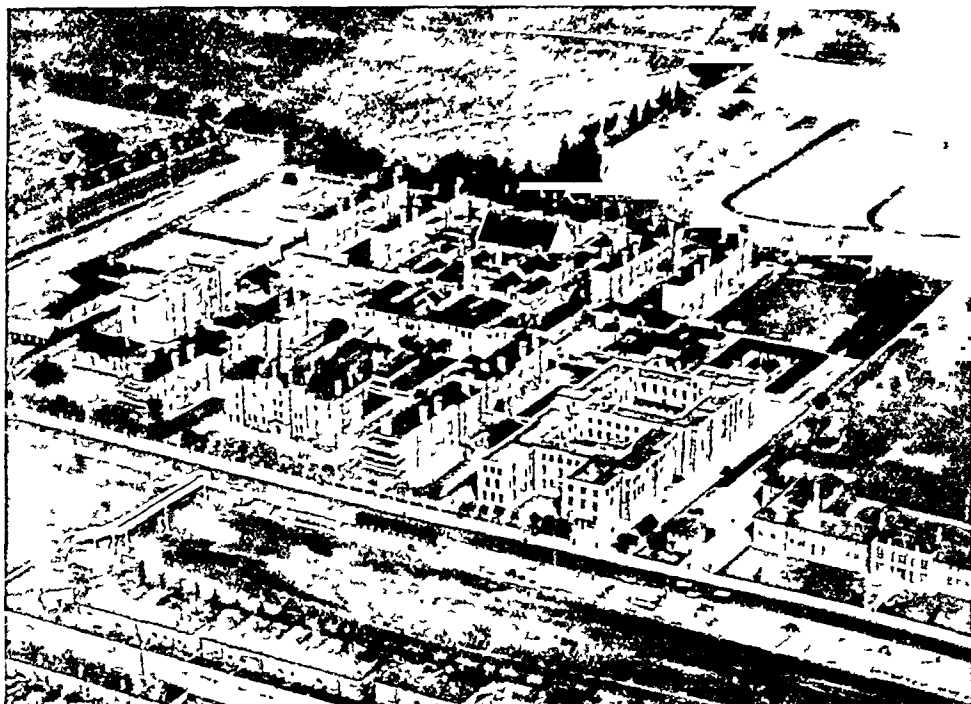


FIG 405—An air view of the Post-graduate School and Hospital.

It would be outside the scope of the present article to attempt any detailed description of the buildings. The medical school, which is joined to the hospital, contains a large out-patient department, which is also used on one or two days each week as a fracture clinic ; two operating theatres, with attached rooms for sterilizing and anæsthesia ; a very extensive unit of pathology, including an animal experiment department, museum, and post-mortem room ; a library ; and an X-ray department for diagnosis and treatment. (*Figs. 405-408.*)

It was appropriate that the first official visit of the Moynihan Surgical Club to London on Oct. 9 and 10, 1936, should have been to the clinic of Professor Grey Turner, because he was one of the original members of the club, which he had entertained previously at Newcastle-on-Tyne, before his migration to

London, where he holds the onerous and responsible post of the first Director in Surgery of the new British Post-graduate Medical School.

Having been welcomed by the Dean of the School, Colonel A. H. Proctor, D.S.O., I.M.S., we began our visit by watching Prof. Grey Turner do three operations. Before each operation he gave a short discussion, together with a typed history of the patient's illness.

Case 1. A Girl of 5 years with Extroversion of the Bladder.—The child had great dilatation of both ureters, highly infected urine, and had had several febrile attacks, which had not been relieved by ureteral catheterization. At a previous operation the right kidney had been drained, with very marked improvement of the



FIG. 406 —The library.

general condition. The abdomen was opened by a midline incision. Both ureters were found to be greatly dilated to a diameter of about 1 in., the right being slightly the larger. The presacral nerve was exposed and resected, with the object of reducing the dilatation of the ureter. It was not considered wise to attempt to transplant the ureter at this stage, and the operation concluded by drainage of the pelvis of the left kidney. The patient stood the operation very well and did not appear to be appreciably upset.

Case 2. A woman of 60 with Recurrent Gall-stone Attacks.—Gall-stone attacks since 1917. In 1931 she suffered from acute cholecystitis, for which the gall-bladder had been drained after removal of the stones. Since April, 1936, she had suffered from recurrent attacks of colic and vomiting, and during each attack she could feel a lump at the site of the operation scar, and she was mildly jaundiced. In August, 1936, she had an abscess of the gall-bladder pointing; this was incised,



FIG 407—An operating theatre

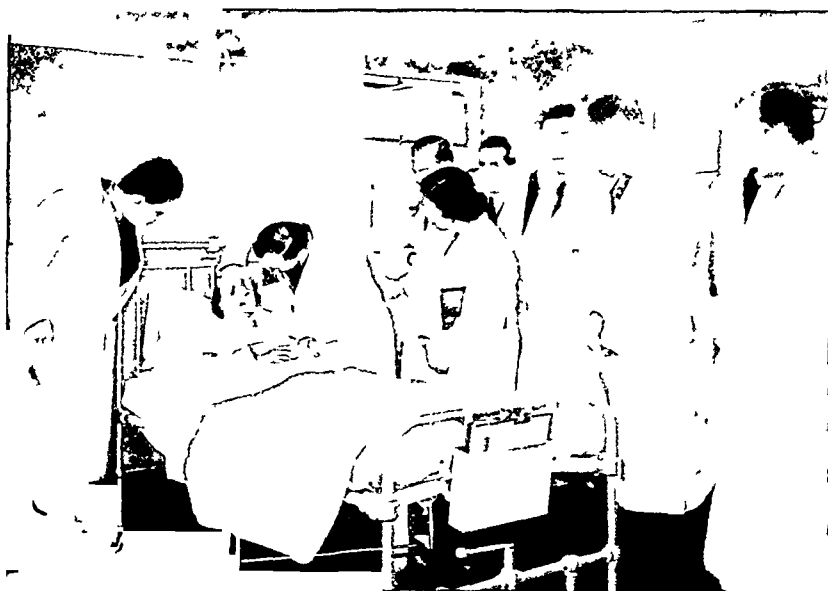


FIG 408—A hospital ward.

letting out purulent bile, but no stones. After this, whenever the sinus closed, there was recurrence of pain and jaundice. The heart showed senile myocardial degeneration with some fibrillation. Lipiodol injection into the sinus showed dilatation of the common bile-duct. The abdomen was opened by Kocher's incision. There were many adhesions between the gall-bladder, anterior abdominal wall, large intestine, and stomach. These were cut through and the gall-bladder and common duct fully explored. The common duct was very distended and contained a gall-stone. It was opened and the stone removed; a sound passed easily into the duodenum. The incision in the common duct was closed and a cholecystectomy performed. The abdomen was closed round a rubber tube draining the region of the common duct. The liver looked as if it were the seat of fatty degeneration. The stone removed was oval in shape and about $1\frac{1}{2} \times \frac{3}{4}$ in.

Case 3. A man of 36 with Pyloric Stenosis: Gastro-enterostomy and Gastrostomy.—Epigastric pain and tenderness for fifteen years. Vomiting began nine months ago and had increased in frequency and in amount. Marked loss of weight. Visible peristalsis from left to right over the umbilical region. X rays had shown great dilatation of a hypotonic stomach. After twenty-four hours the stomach still contained a large residue. Before the operation the stomach had been washed out daily for four days. One pint of intravenous glucose solution was given, and the abdomen opened by a midline supra-umbilical incision. The stomach was found to be greatly dilated and there was a cicatrizing ulcer in the first part of the duodenum. A posterior gastro-enterostomy and a Kader-Senn gastrostomy were performed. The abdomen was closed round the gastrostomy tube.

The stomach was not only very large, but was apparently atonic and certainly thin-walled, and it was on this account that the gastrostomy was combined with the other operation.

In the intervals between the operations we watched Mr. Feggetter doing a cystoscopy and a diathermy resection of the prostate. Also we had an interesting demonstration by Mr. Watson on some fracture cases, chiefly those of intracapsular fractures of the hip, which had been treated by nailing with the Smith-Petersen nail.

In the afternoon we made a tour of the medical school. Professor Kettle showed us the very well-equipped Department of Pathology. The post-mortem room is so large and well lit as to make a good demonstration and lecture theatre. There are tables for conducting four autopsies at the same time. In addition to the daily post-mortems there is a weekly lecture on selected autopsies performed during the week. The Museum is naturally small at the present moment, but it is beautifully arranged, and the descriptive catalogue gives clinical details, while stained microscopical sections are readily available for study. Biochemistry, bacteriology, and animal experimental work are amply provided for.

Dr. Duncan White showed us round the Department of Radiology. The diagnostic radiography is done in five separate rooms, which are used for general work, and for urological, dental, thoracic, and gastro-intestinal examinations respectively. There are twenty-four viewing boxes, and also all noteworthy or exceptional films are at once reduced to lantern slides, so that at a moment's notice there is always at hand a large number of typical or extraordinary pictures for lectures or demonstrations. There is also an X-ray therapy equipment, to which is to be added a radium department.

Later in the afternoon we were treated to a most fascinating demonstration by Professor Grey Turner on various products of his 'bow and spear'. We followed a short résumé of his pioneer work on cancer of the œsophagus, illustrated by specimens from different cases—we saw those specimens of cancer of the stomach, colon, and rectum which have justified him in regarding malignant disease as 'curable'.

Next morning we had various papers and demonstrations: Mr. Henry explained and carried out an extra-peritoneal method for the radical cure of inguinal hernia; Mr. Elliott Smith (a voluntary assistant), the operation of epididymis ligature for enlarged prostate; Mr. Feggetter gave a paper on diathermic resection of the prostate, which greatly impressed us with the potential dangers of the method. Mr. Watson demonstrated specimens from a case of fat-embolism, and we hope to publish this case at an early date. Dr. Aitken (Reader in Medicine) showed us the new type of flexible gastroscope, and we were able to see a gastro-jejunal ulcer on a gastro-enterostomy stoma quite clearly.

On leaving the British Post-Graduate School our feelings of enthusiasm for this great advance in medical education were mingled with those of envy—envy of the staff who have the honour of initiating this great venture, and envy of the post-graduate students who have facilities for seeing and learning those wonders of modern medical science which were not dreamed of when we were young.

So far as it is "the first step which counts", the British Post-Graduate Medical School has already achieved a notable success, and is assured of a brilliant future.

EXPERIMENTAL SURGERY

TOXIN FORMATION IN BURNED TISSUES

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EXTENSIVE burning injuries in man give rise to severe systemic disturbances which change in nature, and evidently also in causation, as time elapses after the infliction of the injury; thus the clinical course of an extensive burn may be divided into several more or less distinct stages. A convenient division is into five stages: (1) Initial shock; (2) Secondary shock; (3) Acute toxæmia; (4) Septic toxæmia; and (5) Healing. The clinical features of each stage have been described and discussed in previous publications.^{10, 11} We are concerned here only with the third stage of 'acute toxæmia', which begins between 6 and 50 hours after injury, which bears a close resemblance to other 'toxic' conditions, and which not infrequently ends in death at about 70 hours from the time of the burn.

The etiology of acute toxæmia has not yet been definitely established. Clinical and pathological studies¹¹ strongly suggest that it may be caused by the formation of a toxin or toxins in burned tissues and the absorption of such toxin into the circulation; some evidence from animal experiment can also be quoted in support of this view, which has met with general acceptance for many years. Recently, however, the view has been criticized adversely and on grounds which are vital for its acceptance—namely, the experimental evidence for the formation of toxins in burned tissue. It seemed to us that a decision on the question, which has important practical applications, was desirable and that it could be reached only by further investigation. The results of such an investigation are recorded in this paper.

REVIEW OF PREVIOUS EXPERIMENTAL INVESTIGATIONS

A very considerable bulk of experimental investigation on the cause of death after burns has accumulated during the past century. From this we have selected for review a few of the more complete and significant researches of comparatively recent date and relating particularly to the question under consideration.

Avdakoff, in 1876, was the first to report that the blood of a burned animal was toxic to healthy animals. Pfeiffer³ in a convincing series of experiments showed that the urine and blood-serum of burned rabbits became toxic to mice, guinea-pigs, and also to rabbits. The toxic principles had both neurotoxic and necrotoxic actions; a study of their chemical characteristics, however, revealed little more than that they were labile substances with some properties in common with nucleoproteins and

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snake venom. The toxic nature of the blood of burned animals was shown also by Robertson and Boyd.⁴ In addition, they tested material from human cases of burns by its action on guinea-pigs; whole blood and cerebrospinal fluid proved poisonous, while blood-serum and blister fluid were innocuous.

Numerous experiments have been reported which seemed to implicate the burned area as the source of the toxic principles which appeared in the blood. By excision and transplantation of burned skin, Vogt⁹ was able to prevent toxæmia in a burned animal and to produce it in the host. Confirmatory evidence was obtained from parabiosis experiments in which the unburned animal developed toxæmia unless separation was carried out within twelve hours. Robertson and Boyd⁴ investigated the action of extracts of burned skin from human cases and animals; such extracts were lethal to guinea-pigs. Their conclusions were that the toxic substance in burned skin consisted of two portions, one of which was *thermostabile*, diffusible, and neurotoxic, the other *thermolabile*, colloidal, and necrotoxic; chemically it was composed of primary and secondary proteoses.

Although there was a large measure of agreement that toxic substances were elaborated in burned tissue and absorbed into the blood-stream, opinion regarding the nature of the toxins was far from unanimous, and many improbable and even bizarre suggestions were made, frequently without basis of evidence. Little information of value has been gained from attempts at chemical identification and isolation; some facts, however, have been recorded which might be held to point to products of protein cleavage.

The theory of toxin formation and absorption has not, however, been universally accepted. Underhill and his associates, for example, attributed the systemic disturbances of extensive burns mainly to increased concentration of the blood;^{6,7} they also held that absorption of a toxin from burned tissues was highly improbable. Direct evidence against the toxin theory was claimed by Underhill and Kapsinow,⁸ who repeated the experiments, and failed to confirm the results, of Robertson and Boyd. They could discover no significant difference in action between extracts of burned skin and extracts of normal skin, nor between blood from burned animals and blood from normal animals.

Consideration of the above and other literature has led us to the conclusion that the evidence from animal experiment for a specific 'burn toxin' is inconclusive, though there is much that is suggestive. Proof of the toxin theory would require the demonstration and identification of the toxic substances not only in the burned area but also in the circulating blood.

We decided to investigate first the question of toxin formation in burned tissues.

METHODS

The experiments were carried out on rabbits. When excessive heat is applied to the skin of a rabbit there is no blistering, but a considerable quantity of œdema fluid accumulates in the subcutaneous tissues; swelling is obvious at 1 hour and increases up to 24 or 36 hours, after which a slow absorption begins. Very mild degrees of injury by heat are followed by hyperæmia and slight œdema, which disappear in a few days. After exposure to higher temperatures œdema is more conspicuous and lasts for a week or longer; the skin, however, finally regains its normal condition except for a persistent scaliness and sometimes a few small isolated

areas of necrosis. Following still more severe injury, the affected skin dies and is finally thrown off as a dry, wrinkled, black slough; eventually such areas are invaded by micro-organisms. Our object was to produce by the application of heat an accumulation of œdema fluid without necrosis of skin and without injury to internal organs by the direct action of heat.

In rabbits the skin of the abdomen was shaved and cleansed by ether and alcohol over an area comprising about one-sixth of the total skin surface. Under full ether anæsthesia, and with aseptic precautions, an incision was made in the middle line and the skin of the area together with subjacent platysma muscle dissected up as two lateral flaps. Heat was then applied to the deep surface of the flaps, except over a thin strip on either side of the incision, by a very brief exposure to the diathermy current; the active electrode was a flat disc about 1 cm. in diameter. The incision was closed and in most instances the burned area was protected by collodion applied over gauze. In other experiments the injury was effected by a jet of steam applied to the external skin surface; by either method an injury of the required severity was obtained. The animals subsequently showed no special untoward symptoms.

At varying intervals after burning, the œdema fluid which had accumulated was collected in the following way. The animal was anæsthetized, the area cleansed with ether, the skin and œdematous subcutaneous tissue dissected off, leaving behind a vertical strip on either side of the operative incision, and as much free œdema fluid as possible was collected. Prior to the dissection a small incision was made through sterilized skin and a few drops of œdema fluid were taken for aerobic and anaerobic cultures. When the burned area had been excised the animal was killed. The excised tissue was cut into small pieces and the œdema fluid expressed from it through a Büchner filter with suction attached. Throughout the procedure all precautions were taken against bacterial contamination of fluid and tissue. It was possible to obtain by this method from one rabbit 10 to 20 c.c. of œdema fluid which, on account of hæmoglobin content, was straw-coloured or pink and which contained also small particles of tissue. The solid particles could then be removed by centrifugation.

The toxicity of œdema fluid was tested by intravenous injection (into an ear vein), by subcutaneous and intraperitoneal injection in unanæsthetized rabbits, and by intraperitoneal injection in mice.

RESULTS

Summary of Results of Intravenous Injection of Œdema Fluid in Healthy Rabbits.—*Table I* shows the results of injecting œdema fluid removed at different intervals from burned areas.

The quantities injected were from 2 to 20 c.c.; 5 to 10 c.c. was an average dose. It will be seen that after a certain period following burning the œdema fluid became highly toxic, and that the development of toxicity was gradual. At forty-eight hours the fluid was frequently lethal.

The Rôle of Bacteria.—In earlier experiments cultures were made from the fluid after removal of tissue and extraction of fluid; in these circumstances a profuse growth of organisms was obtained in both aerobic and anaerobic cultures. When, however, the method of taking cultures through a small incision was employed the cultures proved almost always negative. This was the case also when injury was produced by steam. It was clear that as a rule no organisms were present in the œdema fluid before removal of the burned tissue, and that any organisms in the

fluid used for injection had been introduced during the procedure of extracting the œdema fluid. Moreover, a final survey showed no significant difference in toxicity of œdema fluid whatever the results of cultures. We have, therefore, reported in *Table I* experiments irrespective of the results of bacteriological cultures.

Table I.—RESULTS OF INTRAVENOUS INJECTIONS OF OEDEMA FLUID

INJECTIONS	TOTAL ANIMALS	DIED WITHIN 10 DAYS	DIED AFTER 10 DAYS	ILL BUT SURVIVED	NOT OBVIOUSLY AFFECTED
A. Oedema fluid removed at :—					
4 hours	6	0	0	0	6
6 hours	2	1	1	—	—
12 hours	2	2	—	—	—
24 hours	6	2	—	2	2
27 hours	2	1	1	—	—
48 hours	44	38	2	3	1
B. Oedema fluid removed at 48 hours and passed through Seitz filter	15	7	—	—	8

When the fluid was passed through a Seitz filter to remove organisms, there was usually a diminution in toxicity; toxicity, however, was not abolished (*Table I*). Diminution in toxicity occurred irrespective of the results of cultures made previous to filtering, and was apparently not due merely to removal of organisms.

We have concluded that the development of toxicity of œdema fluid was independent of the growth of organisms in the burned area.

Control Experiments.—œdema fluid removed at four hours after injury was invariably innocuous even in large doses. These experiments served as a control series. In addition, control experiments were done by injecting saline emulsions of large areas of skin immediately after burning, and saline emulsions of normal skin. In no case was any toxic action observed. In both control series injections were made, without deleterious effect, when previous centrifuging of the samples had been omitted. Thus it was shown that the toxic effects of œdema fluid were not merely a result of embolism by tiny particles of tissue. A further possibility had to be eliminated. Roome and Wilson⁵ found that œdema fluid from the traumatized muscles of a limb, on injection into healthy animals, caused immediate death by extensive intravascular clotting. We have not observed intravascular clotting after injecting œdema fluid from burned areas.

Properties of œdema Fluid Removed at Forty-eight Hours after Burning.—It was found that at forty-eight hours the amount of fluid present was nearly maximal and that usually a high degree of toxicity had developed. After this time the risk of bacterial invasion of the œdema fluid became steadily greater. The properties of fluid removed at forty-eight hours were therefore investigated.

Effects of Injection.—Samples of fluid differed in toxicity, and there was also a considerable individual variation in susceptibility of animals injected. Sometimes death occurred within a few seconds after 3 to 5 c.c. had been injected; in such cases generalized convulsions supervened, respiration failed, and the heart stopped. In other instances the animal, after several minutes, developed signs of muscular

weakness, laboured respiratory movements, increased heart-rate, pallor of the ears, and dilatation of pupils; sometimes urine and faeces were voided. The weakness became gradually more marked, the rectal temperature fell after a

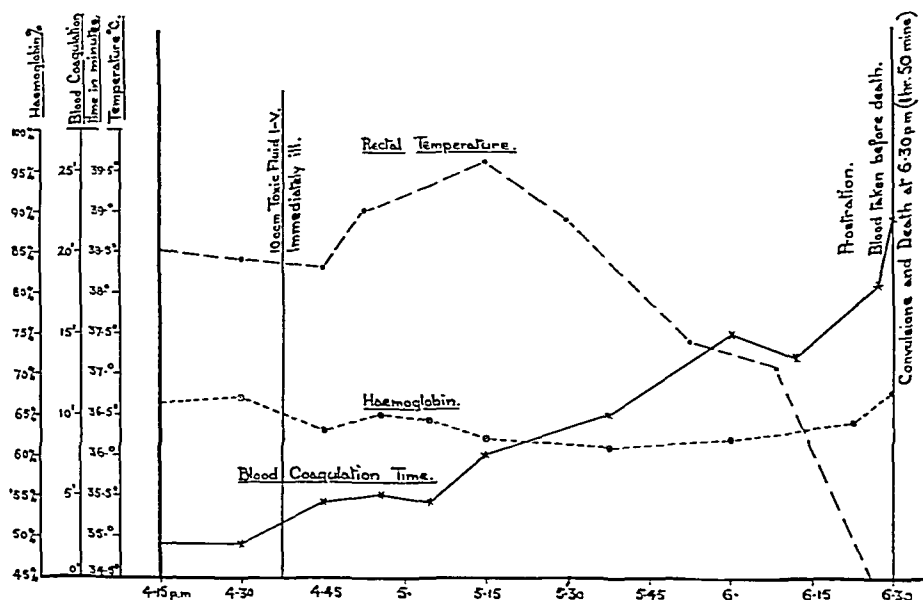


FIG. 409.—Chart showing changes in blood concentration, rectal temperature, and blood coagulation time after intravenous injection of toxic fluid.

FIG. 410.—Effect of intravenous injection of toxic fluid on the blood-pressure. At D, Injection of 1 c.c. of fluid. Fall of pressure from 110 mm. Hg to 56 mm. Hg in 2 minutes. Time marker, 2 sec.

preliminary rise, and death occurred from one to five hours after injection, sometimes preceded by muscular twittings or generalized convulsions. Blood withdrawn from an ear vein showed no alteration in haemoglobin content; the

blood coagulation time was usually increased. *Fig. 409* illustrates the changes in a typical experiment.

Samples of fluid which caused death within a few hours were tested by injection in rabbits under urethane anaesthesia (1.0 to 1.5 g. per kilo); the blood-pressure was

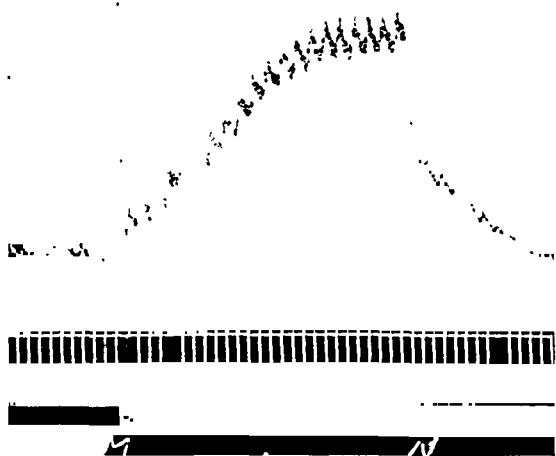


FIG. 411.—Effect of clamping aorta on the hypotension produced by toxic fluid. M to N, Descending aorta clamped. Blood-pressure rose from 30 mm. Hg to 112 mm. Hg. Time marker, 2 sec.

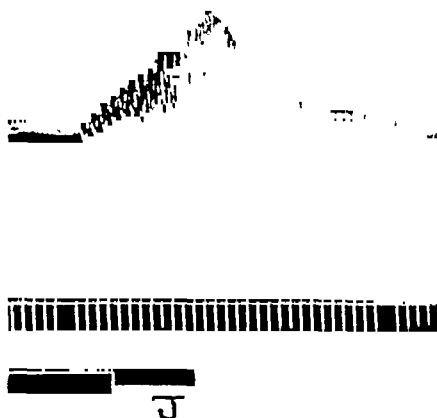


FIG. 412.—Effect of asphyxia on the hypotension produced by toxic fluid. At J, Obstruction of trachea. Blood-pressure rose from 64 mm. Hg to 104 mm. Hg. Time marker, 2 sec.

recorded and a tube tied in the trachea. The blood-pressure sometimes fell immediately after injection and the fall was permanent (*Fig. 410*); in other instances the fall was very slow but progressive. During the collapse of arterial pressure the

thorax was opened under artificial respiration; the heart was beating vigorously though rapidly, and the great veins and the right heart were not dilated. Clamping the thoracic aorta for a short period produced a rise in blood-pressure to the original level (*Fig. 411*). Clearly the hypotension was not due to a primary failure of heart action. Nor was it due to exhaustion of the vasomotor centre, since this responded to temporary asphyxia by a distinct rise in blood-pressure (*Fig. 412*).

In the majority of experiments death occurred at much longer intervals than those mentioned above. The average period of survival of animals which died within ten days, excluding those which succumbed immediately, was calculated as thirty-nine hours. This figure is probably higher than the true period, since in a number of instances death had occurred some hours before it was noted. Usually no change was evident for some hours after injection; in one, two, or more days the animal began to refuse food, became gradually prostrated, and died. In a few cases the animal was immediately ill and remained so for a number of hours but ultimately recovered; in others, and these were numerous, a period of apparent recovery was followed by relapse and death after several days.

Subcutaneous and Intraperitoneal Injection in Rabbits.—Oedema fluid proved toxic when injected subcutaneously or intraperitoneally in rabbits; for a lethal effect quantities larger than those which were used in intravenous injection were required.

Intraperitoneal Injection in Mice.—The quantities injected were 0.5 to 1.0 c.c. The results were closely parallel with those of the same samples in rabbits. The action of the fluid was sometimes striking; death occurred at twenty-four to forty-eight hours, and was preceded by prolonged spasm of certain muscle groups, so that the head was flexed, the back arched, and the limbs and tail extended. Control inoculations of blood and serum from normal rabbits were negative.

Post-mortem Changes.—In rabbits which succumbed immediately no special changes were observed; there was no evidence of bronchial constriction, and, as already stated, intravascular clotting was not found. In animals surviving for a few hours or longer the liver was the organ which sustained particular damage; abnormalities in the kidneys, gastro-intestinal tract, brain, heart, lungs, or suprarenals were slight or absent. The liver in a typical case was pale, yellowish, sometimes of a 'nutmeg' appearance, and abnormally firm in consistence. Microscopic section showed fatty degeneration, which was confirmed by appropriate staining, involving mainly the cells of the centres of the lobules (*Figs. 413, 414*). The liver damage was most severe in animals which died during the first few days; if the survival period were more than a week the changes were less constant and less conspicuous.

The Nature of the Toxins.—The gradual development of toxicity of oedema fluid in the absence of bacterial growth strongly suggested that autolysis of injured tissue was responsible for toxin formation. The possibility that the toxic principles were products of protein cleavage was therefore tested. Various protein fractions were made and injected into rabbits; the results are tabulated in *Table II*.

The albumin fraction was separated from the globulin by appropriate saturation with ammonium (or magnesium) sulphate and the sulphate ions were removed by dialysis through a collodion membrane. The globulin fraction separated out during dialysis into euglobulin and soluble pseudoglobulin fractions. In some cases the two globulin fractions were injected separately. The results showed that the

toxic principles were present in both albumin and total globulin fractions, but were probably associated mainly with the globulin fraction. Alteration of the *pH* so as to secure more complete precipitation of globulin did not affect the toxicity of the albumin fraction, and there was no significant difference in toxicity of the



FIG. 413.—Fatty degeneration of liver-cells. Animal died six days after injection of toxic fluid.

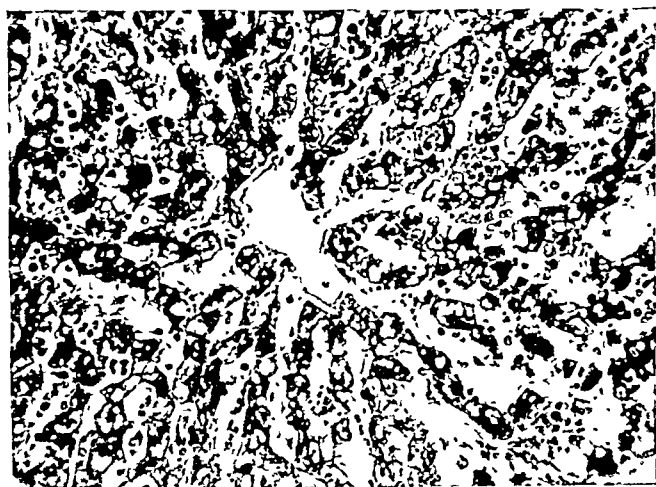


FIG. 414.—Fatty degeneration of liver-cells. Animal died 17 hours after injection.

two globulin fractions. The mother liquor after removal of protein fractions was non-toxic.

Previously it had been shown² that trichloroacetic acid extracts of burned skin, made by the method of Chang and Gaddum,¹ contained an unidentified depressor

substance; extracts of normal skin of rabbits, however, contained the depressor substance in equal or greater amounts. In the present investigation trichloracetic acid extracts of œdema fluid were injected into unanæsthetized rabbits in quantities corresponding to several lethal doses of crude fluid. In one case, where a large dose was injected, death occurred. However, the extract of normal skin and an equal quantity of fluid from a blank extraction also proved fatal. It was therefore impossible to attribute death following administration of trichloracetic acid extract of œdema fluid to any constituent of the fluid resulting from burning. The negative results of the remaining injections suggested that the toxic principles of œdema fluid were not present in such extracts.

Boiling œdema fluid or heating it at 60° C. for 30 minutes lessened the toxicity (*Table II*).

Table II.—RESULTS OF INJECTIONS OF CHEMICAL FRACTIONS

INJECTIONS	TOTAL ANIMALS	DIED WITHIN 10 DAYS	DIED AFTER 10 DAYS	ILL BUT SURVIVED	NOT OBVIOUSLY AFFECTED
Albumin fraction ..	9	5	1	—	3
Total globulin fraction ..	12	9	2	—	1
Euglobulin fraction ..	3	1	—	—	2
Pseudoglobulin fraction ..	4	1	—	—	3
Mother liquor	3	—	—	—	3
Trichloracetic acid extracts	5	1	—	—	4
Toxic fluid after boiling ..	4	1	1	—	2
Toxic fluid after heating at 60° C. for 30 min. ..	2	1	—	—	1
Toxic globulin fraction after boiling	3	1	—	1	1
Toxic albumin fraction after boiling	1	—	—	—	1

The Rôle of Enzyme Action.—In order to test whether toxicity could be produced in œdema fluid when organisms were excluded we carried out the following experiments. Œdema fluid was filtered through a Seitz filter and kept in a refrigerator at 4° C. The fluid, originally non-toxic, proved lethal 10 days later, but became innocuous again at 100 days. Similarly, filtered non-toxic fluid when incubated at 37° C. was strongly toxic at 10 days but had lost toxicity by 30 days. The results suggested that toxin production was by enzyme action. Bacterial growth would presumably have led to a sustained level of toxicity.

Attempts to immunize rabbits by repeated injection of small quantities of toxic fluid proved unsuccessful.

DISCUSSION

In these experiments it has been demonstrated that œdema fluid extracted from a burned area is toxic to healthy animals of the same species. By injecting œdema fluid itself, we avoided fallacies connected with methods of chemical extraction; other fallacies such as the effects of embolism or of introducing hæmocoagulants into

the circulation were also ruled out. The development of toxicity was slow, and, although the experiments were not sufficiently numerous to indicate it clearly, probably steady and progressive, up to forty-eight hours at least. Fluid at four hours after injury was not toxic. Clearly the toxic principles were not formed immediately by the action of heat. We were able to exclude with reasonable certainty the action of micro-organisms and to furnish also some direct evidence that toxic substances could be formed in oedema fluid by enzyme action after organisms had been removed by filtering. In view of these facts it seemed justifiable to conclude that autolysis of injured tissue was the mechanism of toxin elaboration.

The actions of oedema fluid indicated that more than one toxic substance was present. Some samples had a rapidly lethal, apparently neurotoxic, action which was entirely absent from others. Samples which caused death within a few hours did so by virtue of some component which had many points of community with histamine and other capillary poisons. Histamine itself, however, was not then the main toxic element, since, among other reasons, the fluid caused a marked fall in blood-pressure in the anæsthetized rabbit. When, as in the majority of experiments, oedema fluid caused death after a considerable number of hours, there was sometimes little to indicate its mode of action; the chief, though not invariable, effect was to produce degenerative changes in the liver cells. The action of the toxic principles requires further investigation.

In respect of identification of the toxins, the study is also incomplete. The toxic bodies were associated mainly with the globulin fraction, though the albumin fraction was only slightly less implicated, and thus were brought into the category of protein derivatives. The evidence given by the action of trichloroacetic acid extracts, though complicated by the results of control experiments, may be considered as an indication that the more potent of the poisons were derivatives of a higher order than amino-acids or lower peptides. Even if this evidence be disregarded, that from the injection of mother liquor and from the effect of boiling or heating oedema fluid still points to the same conclusion.

The burned animals were in most instances very little affected by the injury during the period of survival allowed, i.e., forty-eight hours—a feature which might be regarded as an objection to the thesis of toxin formation and absorption. In our opinion, however, the objection is not serious. Since the burned area was of limited extent, and the development of toxicity was gradual, the quantity of toxin absorbed during the period of survival would, it is reasonable to expect, be small. Moreover, the capacity of animals to survive repeated small doses of toxin was proved in experiments on attempted immunization. We do not intend to discuss here the question, raised by Underhill and others, of the rate of absorption from burned areas; this feature has not been investigated, and, furthermore, its importance in relation to the toxin theory must rest principally upon the conditions of absorption in human beings.

A comparison of the toxic effects in animals of injected oedema fluid with the clinical condition of acute toxæmia is of special interest. In many respects there is a marked similarity; the resemblance is perhaps strongest in the post-mortem changes, because the striking and constant feature of fatal toxæmia is necrosis and fatty degeneration of the liver-cells in the centres of the lobules. Even without these points of similarity the demonstration of toxin formation in burned areas is in itself suggestive that the action of circulating toxins may play an important part

in the systemic disturbances of burns. Taken in conjunction with evidence, which will be detailed in a separate paper, that, in human cases, increased concentration of the blood, early bacterial infection, and changes in blood chemistry are not essential causes of toxæmia, the results of this investigation, although in some respects incomplete and as yet unaccompanied by a demonstration of toxic properties in the blood of burned animals, might seem of particular significance.

CONCLUSIONS

1. Œdema fluid which accumulates in a burned area gradually acquires toxic properties ; when collected at forty-eight hours after burning it is frequently lethal to healthy animals of the same species.

2. The development of toxicity is independent of the action of bacteria ; there is evidence that autolysis of injured tissue is the responsible mechanism.

3. A study of the actions of toxic œdema fluid suggests the presence of more than one toxic component, and that the proportions of the components vary in different samples.

4. The effects produced include toxic action on the nervous system, circulatory depression, and degeneration of liver-cells.

5. The toxic principles are associated mainly with the globulin fraction. The evidence at present available indicates that the more active principles are higher protein derivatives.

6. The relation of these results to the etiology of acute toxæmia of burns in human cases is mentioned. Features which require further investigation are indicated.

We are indebted to Dr. C. P. Beattie, Bacteriology Department, University of Edinburgh, for assistance and advice in the bacteriological part of the work. The expenses were in part defrayed by grants from the Earl of Moray Fund, University of Edinburgh.

REFERENCES

- ¹ CHANG, H. C., and GADDUM, J. H., *Jour. of Physiol.*, 1933, lxxix, 255.
- ² HARRIS, H. N., WILSON, W. C., and STEWART, C. P., *Proc. Soc. Exper. Biol. and Med.*, 1935, xxxii, 913.
- ³ PFEIFFER, H., *Virchow's Arch.*, 1905, clxxx, 367.
- ⁴ ROBERTSON, B., and BOYD, G. L., *Jour. of Lab. and Clin. Med.*, 1923, ix, 1.
- ⁵ ROOME, N. W., and WILSON, H., *Arch. of Surg.*, 1935, xxxi, 361.
- ⁶ UNDERHILL, F. P., et al., *Arch. of Internal Med.*, 1923, xxxii, 31.
- ⁷ UNDERHILL, F. P., KAPSINOW, R., and FISK, M.E., *Amer. Jour. Physiol.*, 1930, xc, 302, 315, 325, 330, 334, 339.
- ⁸ UNDERHILL, F. P., and KAPSINOW, R., *Jour. of Lab. and Clin. Med.*, 1931, xvi, 823.
- ⁹ VOGT, E., *Zeits. f. exper. Pathol. u. Therap.*, 1912, xi, 191.
- ¹⁰ WILSON, W. C., *Med. Research Council, Spec. Rep. Ser.*, No. 141, 1929.
- ¹¹ WILSON, W. C., *Edin. Med. Jour.*, 1935, xlii, 177.

SHORT NOTES OF RARE OR OBSCURE CASES

ILEOCOLIC INTUSSUSCEPTION OF INFANCY TENDING TO NATURAL CURE

BY JAMES COOK, BIRKENHEAD

THE following case of ileocolic intussusception is of interest, presenting as it does some unusual clinical features. It also illustrates the manner in which the critical period may be tided over pending complete separation of the intussusceptum in cases of 'natural cure'. That a relatively formidable operation was well tolerated by one of such tender years was due to the relief secured from obstruction by the formation of a fistula between the intussusceptum and intussusciens prior to the operation.

HISTORY.—A. L., a female aged 13 months, was admitted to Birkenhead Municipal Hospital on June 15, 1936, having had attacks of vomiting daily for six days. On June 12, following a constipated motion some blood 'oozed' from the anus; thereafter the bowels remained confined until June 14, when a sharp attack of diarrhoea occurred, the stools being dark in colour. There had been several attacks of vomiting on the morning of admission and the bowels had not then moved for twenty-four hours. The infant was a first child, had been bottle fed, and had had no previous illnesses.

ON EXAMINATION.—The infant was well nourished, was somewhat fretful, and had flushed cheeks. The teeth were good, and no abnormalities were found in the heart or lungs. The abdomen was distended but there was no palpable mass. Rectal examination was negative. Temperature was 98° F., pulse 110, and respiration 22. A soap-and-water enema resulted in a dark, fluid offensive motion. There was considerable mucus present and much flatus was passed.

June 16.—Abdominal distension was much less marked. No mass was palpable. Bowels did not open. There was no more blood noted on rectal examination. Vomiting occurred thrice during the night. T. 100°; P. 120; R. 28.

June 17.—A little greenish fluid was vomited. A normally coloured motion was passed. T. 97°; P. 118; R. 26.

June 18.—There was no more vomiting. Spasms of abdominal pain occurred. The abdomen remained distended. A normal motion was passed in the morning. T. 97.4°; P. 120; R. 26.

June 19.—Abdominal distension was less marked. A formed motion was passed. The examination for occult blood was positive. There was no further vomiting. T. 100°; P. 120; R. 28.

June 20.—Abdomen still distended. Stools of normal consistence passed. Occult blood positive. T. 98°; P. 124; R. 28.

June 21.—There was no vomiting, rigidity, or apparent tenderness. Abdominal distension persisted. Rectal examination was negative. Bowels were open thrice, the motions being relaxed and offensive. T. 98.8°; P. 118; R. 28.

June 22.—Abdominal distension more marked and visible peristalsis noted for first time. Vomiting recurred. There was offensive diarrhoea, and blood and mucus were passed per rectum. T. 103.8°; P. 150; R. 34.

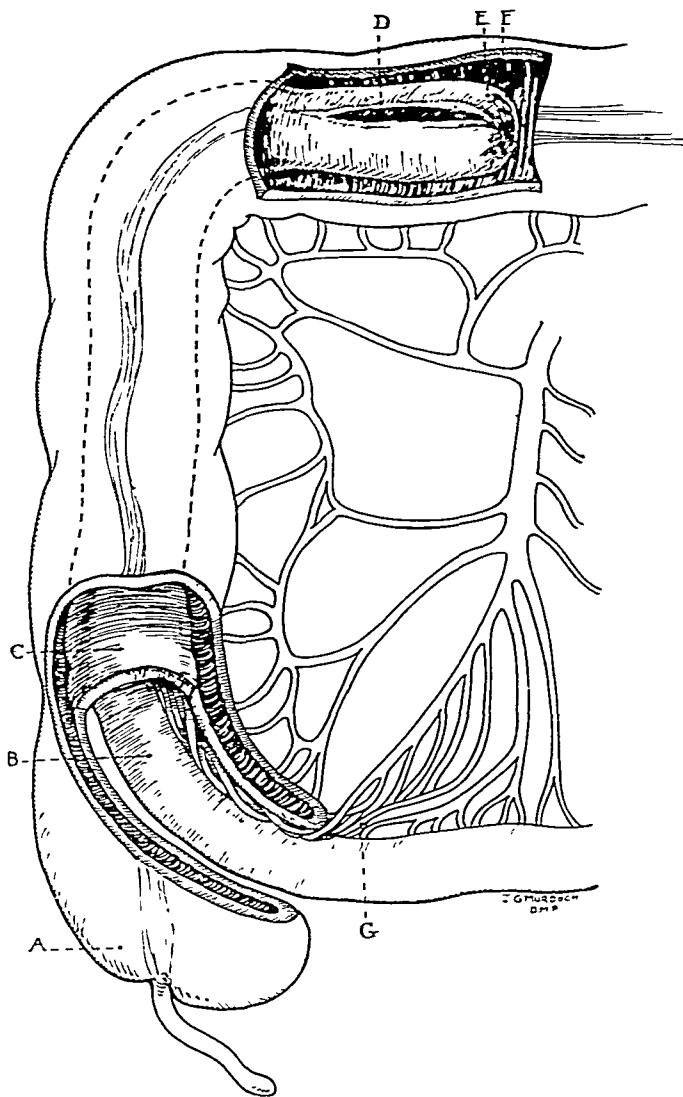


FIG. 415.—Diagrammatic representation of appearances found on opening abdomen. A, Cecum with appendix; B, Entering layer of intussusceptum; C, Returning layer of intussusceptum; D, Fistula between ileum and transverse colon; E, Apex of intussusception; F, Lumen of ileum occluded by inflammatory edema; G, Indrawn mesentery.

This was the 13th day of the child's illness, yet her general condition remained fairly satisfactory. Emaciation was absent. There had been no vomiting from June 16 to June 22, and formed motions were passed on the 8th, 9th, and 10th

days of the illness. The recurrence of vomiting together with the appearance of visible peristalsis on June 22 called for abdominal section.

OPERATION.—Under chloroform and ether anæsthesia the abdomen was opened through a right paramedian incision and an intussusception of the ileocolic type

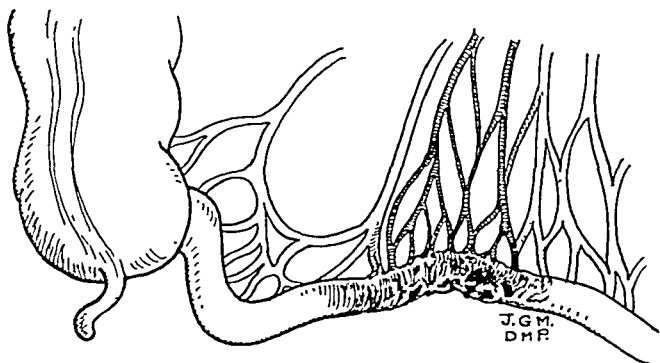


FIG. 416.—Illustrates the appearances after reduction of the intussusception. The gangrenous part of the ileum, its perforated portion, and the thrombosed mesenteric vessels are shaded.

was found, the apex reaching to the middle of the transverse colon (*Fig. 415*). Reduction was carried out with especial care. There was a considerable amount of plastic lymph between the two serous layers of the intussusception, and when reduction was completed it was found that the apex had sloughed so that a fistula had existed between the ileum and the transverse colon. The mesenteric vessels in relation to the necrotic portion of the ileum were thrombosed (*Fig. 416*). The intussusciens was relatively healthy. The appendix was not involved in the process and was not removed. The affected portion of the ileum was resected and an end-to-side anastomosis of ileum into ascending colon was carried out (*Fig. 417*). A rubber tissue drain was passed down to the site of the anastomosis and the abdomen closed in layers.

POST-OPERATIVE COURSE.—On the evening of operation the temperature reached 103.3° F., remained elevated for six days, and then returned to normal. The bowels were opened three to four times daily following the operation, the motions being somewhat offensive for the first two days. At no time did the child's condition after the operation cause concern. There was no recurrence of vomiting, sterile water and later glucose water and Benger's food being well tolerated.

COMMENT.—The case is one of ileocolic intussusception in a female infant. Most children suffering from this disease die within the first week if not operated upon, yet this child was admitted to hospital on the sixth day and survived a severe abdominal operation on the 13th day of her illness. It is estimated that approximately 2 per cent of cases not operated upon may survive, 'natural cure' following

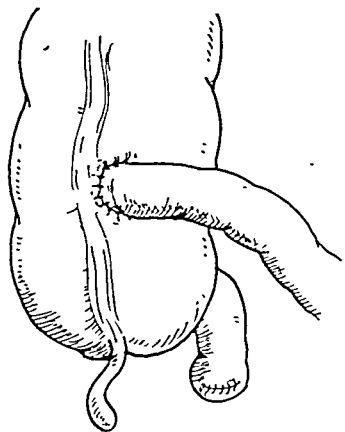


FIG. 417.—Represents appearance after resection and end-to-side anastomosis.

sloughing, separation, and expulsion of the intussusceptum. The sloughing probably commences at the apex of the intussusceptum, and it is most likely that in those cases surviving, nature first establishes (as in this case) drainage of the obstructed bowel. Indeed it would appear that in this case such drainage had been established prior to admission to hospital. Offensive stools in the absence of a palpable abdominal mass, together with a swinging temperature, gave rise to the erroneous diagnosis of primary ileocolitis. The infection actually present in this case arose in all probability from the gangrenous process present in the intussusceptum.

The author wishes to thank Dr. J. G. Murdoch for the accompanying illustrations.

A CASE OF MULTIPLE MYELOMA

BY LIEUT.-COLONEL M. M. CRUICKSHANK, I.M.S., MADRAS, INDIA

THIS case was one in which unilateral proptosis due to an intra-orbital tumour developed in the course of a year in association with X-ray appearances in the skull indicative of isolated areas of rarefaction. The X-ray appearances were not unlike those seen in skeletal lipoid granulomatosis, the Schüller-Christian syndrome. The patient was referred to the Ophthalmic Hospital, Madras, for opinion, where Col. R. E. Wright performed tarsorrhaphy, explored the orbit, received a pathological report of a round-celled sarcoma, and treated the intra-orbital growth with radium implantation. The mass disappeared after 864 mg.-hr. had been given, and, as it was considered that nothing further could be done in view of the fact that the condition was probably multiple myeloma, a diagnosis supported by a later histopathological report, the patient was returned to Madura. An X-ray print shows the condition of the skull. Eventually the whole skeleton showed similar invasion. The detailed notes of the case are as follows:—

G., Hindu, male, age 30.

May 11, 1934.—Seen in the Ophthalmic Department, Government Headquarter Hospital, Madura, complaining of pain in and protrusion of the left eye—duration six months. There was marked proptosis down and out of left eyeball, with definite uniform diffuse swelling, extending temporal-wards from the outer orbital margin, and inwards into orbit, causing proptosis of the eyeball (*Fig. 418*).

R.E.V., 6 6; L.E.V., 6/36.

Pupils equal and active. Fundi—right and left—normal.

All movements in both eyes free except upward in left.

Blood for Wassermann reaction, negative.

Urine: reaction, acid; albumin and sugar, nil; sp. gr., 1010.

July 12.—Patient again seen. Tumour mass had definitely increased in size and a diagnosis of ? sarcoma extending into orbit was made. X rays showed 'honeycombing', a number of small rounded areas of rarefaction, suggestive of the changes seen in the Schüller-Christian syndrome.

Patient sent to Col. Wright for opinion. Col. Wright removed part of the tumour for microscopic examination, the pathologist at first reporting lympho-sarcoma. Tarsorrhaphy of outer third was done, and radium implanted—total dose 864 mg.-hr.

Orbital growth disappeared, and proptosis became reduced to vanishing point. Sept. 3.—Patient again seen. R.E.V., 6/6; L.E.V., 6/60.

I then lost sight of the patient, but Dr. Natesan, Radiologist, Madura, who had been asked to trace him, did so, took a fresh series of X-ray films, and sent

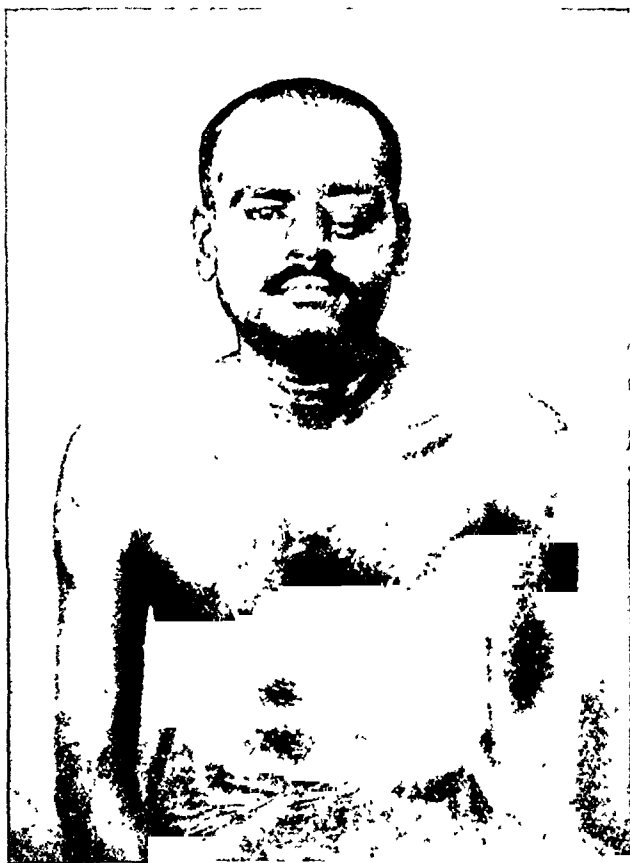


FIG. 418.—Showing proptosis and displacement of the left eyeball.

them to me at the General Hospital, Madras. The films showed an increase of rarefied areas all over the cranium (*Fig 419*).

Nov. 20.—Patient admitted to the General Hospital, Madras, for further investigation. He now complained of severe pain over the right side of chest and back, and the headache, though of less severity than formerly, still persisted. Proptosis of the left eyeball was again present. Patient stated that his general health had been good, though he looked thinner and less well nourished than he did in May, 1934.

Blood Chemistry (investigated by Dr. Mannadi Nayar).—

Blood-sugar	109.6 mg. per 100 c.c.
Blood-lipoid	270.8 " " " "
Blood-cholesterol	181 " " " "

Blood-count.—

R.B.C.	4,650,000
W.B.C.	9375
Polymorphs	66 per cent
Lymphocytes	30 " "
Eosinophils	1 " "
Mononuclears	3 " "

Wassermann reaction, negative; Kahn test, negative.



FIG. 419.—Radiograph of skull.

Radiography.—X-ray examination of the skeleton was carried out by Dr. Pillai, of the Barnard Institute of Radiology. The radiological findings were:—

Chest: There is a tendency to early fibrotic changes in the right lung, chiefly at the upper lobe. Tendency to calcification of the costal cartilages. Hilar shadows rather dense.

Humerus: There is a large area of atrophic changes of the fibrocystic type in the upper half of the left humerus and a small area in the lower end on its external aspect. There are similar changes noted in the whole of the right humerus (Fig. 420).

Scapula: The left scapula shows similar changes at the lower half of the axillary border (Fig. 420).

Radius: The upper end of the left radius shows an area of atrophic changes at the level of the tubercle of the radius. The upper end of the right radius also shows similar changes of an early nature.

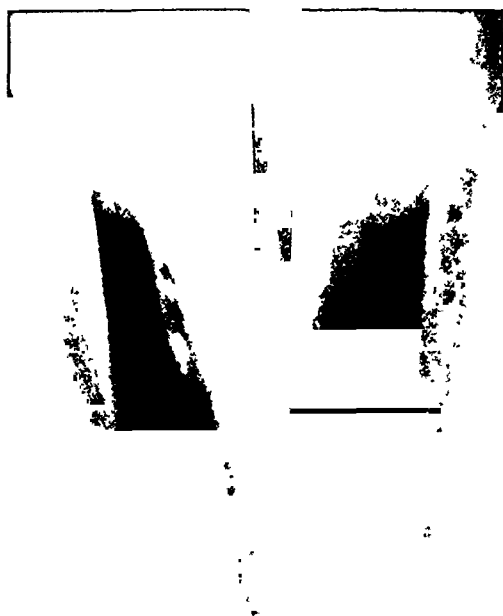


FIG. 420.—Radiograph of humeri and scapulae.



FIG. 421.—Radiograph of pelvis and upper parts of the femora.

Lower limbs: The alæ of the iliac bones, the pubis and the rami, and the upper ends of the femora also show similar changes (*Fig. 421*).

Jan. 25, 1935.—Arrangements had been made to repeat all blood examinations but the patient suddenly left the hospital and proceeded to his home.

June 6.—Dr. Natesan, who had got in touch with the patient, sent the following note from Madura :—

General Condition.—Highly emaciated—"skin and bones"; bed-ridden; puffiness of the cheeks and feet; markedly anæmic; abdomen tympanitic.

Skull.—Left eyeball proptosis as before. A slight softish tumour mass, the size of a bean, has appeared just above the glabella. Probably on account of his emaciation the left parietal eminence appears slightly bigger than normal.

Eye.—Vision in left eye—perception of light; right eye—normal.

Respiratory System.—Slight cough. Wooden dullness over the whole of the chest. ? further deposits in lung.

Urine.—Scanty, high-coloured, loaded with albumin. Bence-Jones reaction—negative.

June 18.—Dr. Natesan reported that the patient died on June 15.

FINAL HISTOPATHOLOGICAL REPORT (Dr. A. N. Goyle).—Myeloma.

Detail of Cellular Structure.—"The cells themselves chiefly conform to two types, one fusiform or ovoid with deep-staining nuclei with indistinct chromatin, and the other with large rounded light-stained nuclei showing well-defined chromatin nodes. These cells show very little protoplasm. Under the oil-immersion lens, the nuclear chromatin is not arranged in a regular cartwheel fashion as in the plasma cell, but there is some resemblance in the nuclear structure to those cells. The protoplasm is scanty and can hardly be made out. It does not show the basophilic character of the plasma cell. Here and there, large cells with large nuclei can be made out. Mitotic figures are very few in the section. I think the dark-staining cells are more or less of the same type, where the nuclei have undergone compression. In some places the nucleolus has taken an acidophilic staining. With Unna Pappenheim, these cells do not show the differential staining of plasm cells, since the protoplasm can hardly be made out. On the whole the nuclear structure is more that of an endothelioid type of cell."

A CASE OF DIAPHRAGMATIC HERNIA

By T. W. MIMPRISS

FROM THE SURGICAL UNIT, ST. THOMAS'S HOSPITAL, LONDON

ALTHOUGH cases of diaphragmatic hernia are now recognized as being comparatively common, this case is recorded as it presents certain unusual features.

The patient was a female child, who was first brought to hospital in February, 1935, at the age of nine months. Breast-feeding had been abandoned after two months, and since then attacks of vomiting had occurred with increasing frequency and severity. Her weight at the age of nine months was 12½ lb. Her diet was carefully adjusted, but the attacks of vomiting continued, and her weight on admission in May, 1935, at the age of 12 months, was only 13½ lb. Large vomits occurred

about twice a day, and the child was only able to take liquids and then very slowly; altered blood was present in both vomit and fæces. No abnormal signs were detected in the abdomen, but a radiological examination after a barium meal showed that part of the stomach was situated in the posterior mediastinum to the right of the vertebral column (*Fig. 422*). After two months' careful dieting in the ward, it was apparent that no improvement had occurred or could be expected.

FIRST OPERATION.—Laparotomy was performed by Mr. Max Page on July 10, 1935, the child then being fourteen months old; the pylorus and the greater part of the stomach were reduced from a hernial sac in the neighbourhood of the oesophageal opening and an attempt was made to repair the opening by catgut

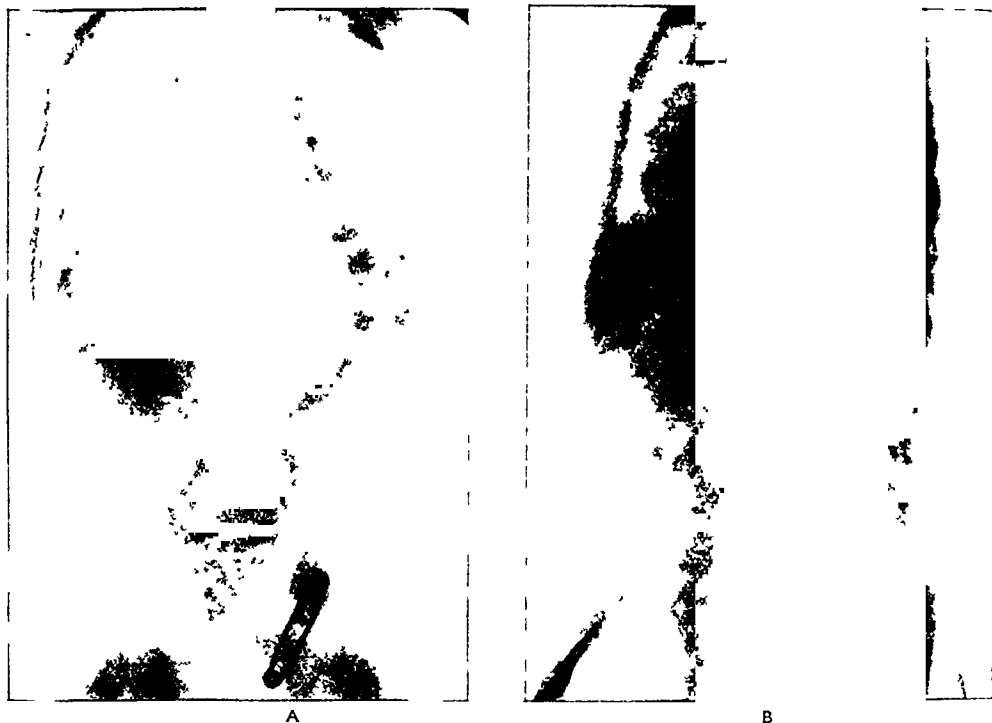


FIG. 422—Radiograph after barium meal, showing the greater part of the stomach in the posterior mediastinum. A, Antero-posterior view; B, Lateral view.

sutures placed across the orifice. The child made a good recovery, vomiting ceased, and weight was put on rapidly. This improvement lasted for five months, although radiological examination one month after the operation showed that the hernia had recurred.

In December, 1935, the child was readmitted owing to recurrence of severe vomiting for four days; the weight was then 19 lb. Radiological examination showed that the hernia was present. Vomiting continued and the child rapidly lost weight.

SECOND OPERATION.—On Jan. 15, 1936, a second laparotomy was performed by Mr. Max Page. The pylorus and the greater part of the stomach were again reduced from the hernial sac with some difficulty; the presence of adhesions

prevented any attempt at repair in so feeble a subject. Gastropexy was performed by suturing the greater curvature of the stomach to the anterior abdominal wall. The child's post-operative condition was poor; vomiting continued, and she died on the third post-operative day.

POST-MORTEM EXAMINATION.—The hernial sac was empty, the stomach was attached to the anterior abdominal wall, and no leakage or obstruction was apparent. The hernial orifice was caused by gross dilatation of the œsophageal orifice in the diaphragm; the œsophagus was short, so that the upper third of the stomach was always above the diaphragm; sections taken from the thoracic portion of the stomach prove that this is stomach and not a dilated œsophagus. The anterior surface of the stomach was attached to the anterior margin of the œsophageal opening, and the sac of the hernia, consisting of the upper part of the lesser sac



FIG. 423.—Diaphragmatic hernia. Hernial orifice seen from below.
A, Pylorus; B, Posterior aspect of stomach.

of the peritoneum, passed upwards behind the stomach into the posterior mediastinum on the right of the vertebral column (*Fig. 423*). Before operation the greater curvature of the stomach and the pylorus had rotated upwards into this hernial sac. The œsophageal opening caused no constriction of the stomach, and the child's symptoms were due to this rotation of the greater curvature and the pylorus through the orifice into the sac.

The œsophageal orifice of the diaphragm, although dilated, had normal boundaries, and using Dunhill's classification this represents an extreme example of the short-œsophagus type of diaphragmatic hernia.

The points of interest are:—

1. The sac was formed entirely from peritoneum of the lesser sac. The anterior wall of the stomach was attached firmly to the anterior margin of the œsophageal opening. There was no sac passing into the thorax in front of the stomach.

2. The sac passed into the posterior mediastinum entirely to the right of the vertebral column.

3. The possibility of gastropexy proving a satisfactory procedure.

In this case the obstructive symptoms were due to rotation of the greater curvature and pylorus through the sac into the mediastinum and not to the fact that the upper third of the stomach was situated permanently in the thorax.

OBSERVATIONS BY MR. C. MAX PAGE.—

The specimen described is unusual in the large size of the hernial sac extending into the right pleural area. Operation became imperative at an early age on account of the severity of the symptoms caused by the herniation of a large section of the stomach. A radical cure of the hernia by the thoracic route did not seem justifiable in this infant. It would, however, appear possible that if a fixation of the stomach had been carried out at the first operation the child might have survived. It seems likely that an effective fixation of the distal half of the stomach would prevent prolapse of sufficient of that viscus into the hernial sac to produce symptoms.

I wish to thank Mr. C. Max Page for permission to publish this case, which was under his care, and to express my indebtedness to Sir Thomas Dunhill for his assistance in interpreting the findings.

AN UNUSUAL VASCULAR COMPLICATION OF CERVICAL RIB

By E. R. FLINT

SURGEON TO THE GENERAL INFIRMARY, LEEDS

NELLIE A., aged 45, was admitted to the General Infirmary at Leeds on May 11, 1936, under Dr. McAdam, who obtained from her the following history.

HISTORY.—Except for poor circulation all her life, in the hands particularly, there was nothing of much note until two months before admission, when she noticed an aching pain in the right thenar eminence; the pain passed into her wrist and palm, and more recently upwards into the shoulder. She had no similar pain in her left arm or anywhere else. During the two months she had slight numbness in the right index finger. For several years she had noticed a small pulsating lump in the right supraclavicular fossa. On Sunday, May 17, it was observed that the pulsation had disappeared—it did not re-appear again—and on the same day the lump became painful and much larger, and the pain in her hand worse.

ON EXAMINATION.—Examination five days before operation showed an ill-defined swelling filling the right supraclavicular fossa; it was tender and fixed deeply, but not to the skin. There was no pulsation in or over the lump. Palpation caused local pain, but no radiation. There was no pulsation in any of the vessels of the right arm; on the left side pulsation was normal. There was slight wasting of the muscles of the arm and hand on the right side and some diminution in muscular power. Sensation was normal and equal on the two sides; the colour of the right hand was rather bluer than on the left side. No other abnormal physical signs were present.

X rays showed a cervical rib on both sides, apparently complete.

Dr. McAdam asked me to see her with a view to surgical treatment.

OPERATION (May 25).—After dividing the skin and platysma (*Fig. 424*) a yellowish-white non-pulsating tumour presented external to the scalenus anticus. It was about the size of, and shaped very like, a Victoria plum, and extended a little

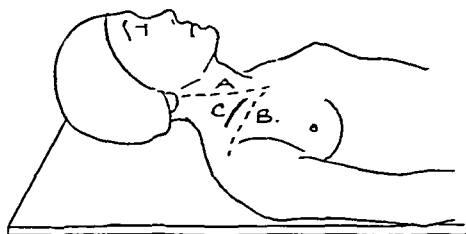


FIG. 424.—Showing position and shape of incision. The sternomastoid is indicated by the broken line A, the clavicle by B; C shows the incision.

beneath the scalene (*Fig. 425*). A small incision was made into it and some clot evacuated. The tumour lay on a cervical rib, which had a keen anterior edge, and at one place a rather sharp projection. The rib was complete; that is to say, it was united to the first rib by bone, and ran a fairly straight course between its vertebral and costal attachments. The scalenus anticus was divided, and the

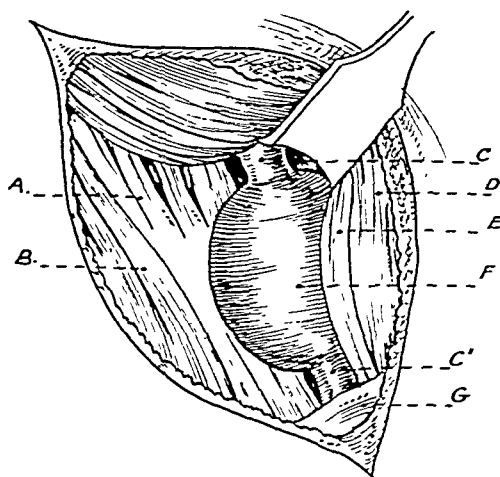


FIG. 425.—Condition found on exposure, with the tumour in situ. A, Brachial plexus; B, Scalenus medius; C and C', Subclavian artery; D, Sternomastoid; E, Scalenus anticus; F, Tumour; G, Clavicle.

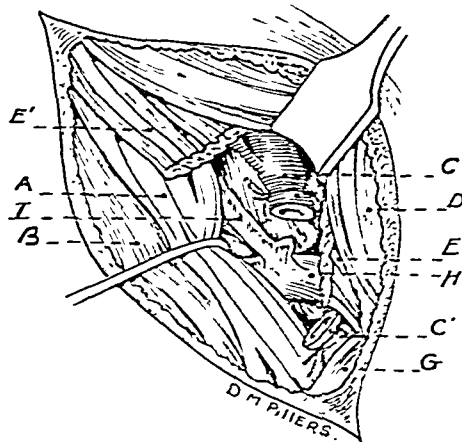


FIG. 426.—After ligature of subclavian artery and removal of tumour. A, Brachial plexus, held aside; B, Scalenus medius; C and C', Subclavian artery ligatured; D, Sternomastoid; E and E', Scalenus anticus divided; G, Clavicle; H, 1st rib; I, Cervical rib (note sharp edge).

subclavian artery was seen entering and leaving the swelling; ligatures were placed above and below the tumour and it was removed, the rib being left in situ (*Fig. 426*). The patient was discharged on June 5 in good condition.

On July 24 the patient was seen again. All pain has gone, and she is doing house-work, which she had been unable to do for several weeks before operation. The hand is a good colour.

PATHOLOGICAL REPORT (Prof. M. J. Stewart).—Sections have been taken both across the rupture in the wall of the artery and across the non-ruptured portion. Actually the breach in the arterial wall does not suggest an aneurysm, but rather a sudden rupture of an arterial wall which had been weakened from without. The lumen is lined by organizing thrombus of some standing, and the middle coat shows much diffuse fibrosis, but the chief changes are in the adventitia and external to it, where there is a broad zone of active granulation tissue. This is in keeping with the suggestion that pressure of a cervical rib may be the prime etiological factor. None of the changes suggest a syphilitic arteritis, although the peri-arteritis might conceivably be syphilitic in nature. This, however, seems unlikely, since the middle coat shows no such change.

Commentary.—Many of the features presented by this case conform to the usual clinical experience: for example, the patient was a middle-aged woman with an accessory rib on both sides (about 75 per cent of cases) but symptoms only on the right side. The unusual thing about this patient is the type of vascular complication, which was apparently a false aneurysm brought about by the constant pressure of an unusually sharp-edged rib. Thrombosis in the subclavian artery has been seen fairly often, and aneurysm has been reported (Billington¹ records a fusiform aneurysm in association with a cervical rib), but I have been unable to find any description in the literature of a case such as is here described.

Two causative factors appeared to be present: (1) The almost cutting edge of the rib; (2) Its nearly straight course, giving the scalenus anticus the fullest opportunity for constricting the artery. Telford and Stopford² find it difficult to believe that pressure is exerted on the subclavian artery in the presence of a cervical rib; the case reported above would seem to make it quite certain that such pressure does occur sometimes, though it is not the cause of all the symptoms.

No attempt was made to remove the rib in my case, as I believe this to be unnecessary and certainly more mutilating than simple division of the scalene at or near its attachment to the first rib; such a division opens up the space at the root of the neck quite enough to accommodate all the structures freely.

REFERENCES

¹ BILLINGTON, W., *Brit. Jour. Surg.*, 1931, xix, 334.

² TELFORD, E. D., and STOPFORD, J. S. B., *Ibid.*, xviii, 557.

THYROID TUMOUR WITH MULTIPLE METASTASES

By JOHN WILLIAMSON

MEDICAL OFFICER, TANGANYIKA TERRITORY

THIS case was seen by me in 1932, soon after W. K. Connell had published a paper in the *BRITISH JOURNAL OF SURGERY*¹ on two cases of thyroid metastases seen by him in Dar-es-Salaam.

I do not think there is any doubt but that this is a case of thyroid metastases, but it differs from Dr. Connell's cases in that the metastases are multiple and the enlargement of the thyroid is very marked.

Unfortunately my notes on the case are not available, and the following very brief history has had to be supplied from memory.



FIG. 427.—Photograph of the patient showing the thyroid enlargement and the growths in the skull.



FIG. 428.—Radiograph of the skull.

The patient was a female African, of about 26 years of age, and she came from a village near Turiani, in the Morogoro District of Tanganyika Territory. She was a healthy individual, and only came to Morogoro Hospital because she was seen by a keen African District Sanitary Inspector, and his description aroused my curiosity. Histories are always difficult in Africans, and this case was no exception to the rule. It would seem fairly certain, however, that the growths had been present for at least ten years, and that the rate of growth was very slow. Some of her friends actually said they could not remember her without them. She was married to a man many years her senior, and had had two healthy children, both of whom died in early childhood. The Wassermann reaction was ++.

There was nothing of note on examination. The pulse was regular in rate and rhythm, was 70 per minute, and responded normally to exercise.

The photograph shows clearly the healthy appearance of the patient (*Fig. 427*), and the radiograph the crater-like condition of the new growths (*Fig. 428*).

I have to thank The Honourable The Director of Medical Services, Dar-es-Salaam, for his permission to publish the illustrations. The radiograph was taken by Mr. J. E. Brunnen, of the Tanganyika Civil Service, and intensified by Messrs. Kodak Ltd.

REFERENCE

- ¹ CONNELL, W. K., "Thyroid Metastases in Bone", *Brit. Jour. Surg.*, 1929-30, xvii, 523.

REVIEWS AND NOTICES OF BOOKS

Techniques Chirurgicales. By A. GOSSET, Chirurgien de la Salpêtrière, Professeur de Clinique chirurgicale à la Faculté de Médecine de Paris; with the collaboration of MM. L.-G. AMIOT, IVAN BERTRAND, JEAN CHARRIER, P. FUNCK-BRENTANO, J. GARCIA-CALDERON, JEAN GOSSET, P. HAUDUROY, R. LEDOUX-LEBARD, R. LEIBOVICI, G. LEWY, P. PETIT-DUTAILLIS, P. ROUCHÉ, R. SAUVAGE, G. SEILLÉ, R. SOUPAULT, M. THALHEIMER, and E. WALLON. Imperial 8vo. Pp. 434 + viii, with 219 illustrations. 1936. Paris: Masson et Cie. Paper covers, Fr. 105; bound, Fr. 125.

HERE M. Gosset and his colleagues present an account of the Surgical Service in the Salpêtrière Hospital in Paris, of which M. Gosset has been the Director for more than twenty years.

In addition to a number of technical articles, e.g., on Diaphragmatic Hernia and Intestinal Obstruction, which describe the activities of the Clinic, Gosset enunciates his matured convictions concerning the housing, organization, and staffing of a surgical unit. The organization comprises both the Clinic of the Faculty and also an Anti-cancer Centre such as exists in Paris and in some of the large French towns. The number of beds in the Clinic is 300, which are divided among pavilions, each containing 30 beds. To each pavilion two assistants are attached who work under the authority of the "chef de service". The Clinic includes numerous assistants, both surgical and medical, and a complete service of radiology, radiotherapy, curie-therapy, and laboratories of pathology, chemistry, bacteriology, and hæmatology. This contribution should be of great service to anyone contemplating the organization of a surgical unit.

Minor Surgery. By FREDERICK CHRISTOPHER, S.B., M.D., F.A.C.S., Associate Professor of Surgery at the Northwestern University Medical School, Chicago; Chief Surgeon at the Evanston (Ill.) Hospital. With a Foreword by ALLEN B. KANAVEL, M.D., F.A.C.S., Professor of Surgery at the Northwestern University Medical School. Third edition, reset. Large 8vo. Pp. 1030, with 709 illustrations. 1936. London and Philadelphia: W. B. Saunders Co. 42s. net.

MINOR SURGERY is defined here as the surgery which has a low mortality; which requires but few assistants; and which is done generally in the hospital out-patient department or in the office.

Though dealing with minor surgery it is not a little book, for it is an octavo volume of 1030 pages. Nor does it treat the subject as one of minor importance. Rather it emphasizes the point that minor surgery may become major surgery through carelessness or incompetence, and that there cannot be too much instruction in minor surgery.

The whole range of the subject is treated most exhaustively and sensibly, and it should prove a useful book of reference to all students beginning surgery and also to resident medical officers.

One of the best sections in the opinion of the reviewer is that dealing with the treatment of wounds and the methods of securing early healing and restoration of function.

Manual of Emergencies: Medical, Surgical and Obstetric: Their Pathology, Diagnosis and Treatment. By J. SNOWMAN, M.D., M.R.C.P.Lond. Third edition. Crown 8vo. Pp. 401 + ix. 1936. London: John Bale, Sons & Danielsson Ltd. 10s. 6d. net.

It is very remarkable how much useful information has been packed into this small manual. It was originally based on the treatise of Lenzmann, but has been well adapted to the modern standard of teaching in British text-books. After treating of shock, the various emergencies which may affect the respiratory, cardiac, nervous, alimentary, and urinary systems are dealt

with. In the abdomen acute general peritonitis is discussed before the causative conditions, e.g., appendicitis, and we think that this is a mistake. Fifty pages are devoted to poisons, and the book concludes with a consideration of obstetrical emergencies. The manual is clearly written and its information accurate and reliable.

Operative Surgery. By ALEXANDER MILES, M.D., LL.D., F.R.C.S. (Edin.), Consulting Surgeon, Royal Infirmary, Edinburgh, and D. P. D. WILKIE, M.D., F.R.C.S. (Edin. and Eng.), Professor of Surgery, University of Edinburgh. Second edition. Demy 8vo. Pp. 631 + xx, with 329 illustrations. London: Oxford University Press. 21s. net.

THIS is the second edition of a text-book on operative surgery, the first edition of which was reviewed in this JOURNAL. The book is intended for students attending a course of operative surgery. It is well illustrated, and sets out in lucid manner all the operations which a student is expected to know.

The Surgical Technic of Abdominal Operations. By JULIUS L. SPIVACK, M.D., Assistant Professor of Surgery, University of Illinois College of Medicine; Professor of Operative Surgery and Surgical Anatomy, Cook County Graduate School of Medicine; etc. 10½ × 7½. Pp. 718 + xvi, with 362 illustrations. 1936. Chicago: S. B. Debour.

IN a volume of some size the author describes with meticulous detail the surgeon's technique within the abdomen. No attempt is made to give indications for the procedures described; he deals with the *How*, not the *Why*. Starting with the simplest procedures of knot-tying and sutures, he builds up the detailed steps of typical abdominal operations. The field is covered very thoroughly, and some of the procedures are of historical rather than of practical interest.

In spite of the limitations necessarily inherent in a book of this type, it has a very definite place in the surgical library, both for the post-graduate student of surgery and for the man of maturer years, and one has only to dip into its pages to realize how relatively narrow a groove of technique one is apt to develop and to hand on to one's assistants. Whilst apprenticeship is essential, it is the richer for the study of surgical technique as a basic factor in the craft of surgery. The illustrations are numerous and give reality to every operative step described in the text, whilst a full bibliography rounds off each chapter.

Appendicitis: When and How to Operate. By W. J. STEWART MCKAY, M.B., B.Ch., B.Sc., Senior Surgeon, Lewisham Hospital, Sydney. Demy 8vo. Pp. 260 + xii, with 16 illustrations. 1936. Sydney: Angus & Robertson Ltd. 12s. 6d. net.

IT is hard to conceive that in these days any medical man, even in the wilds of Australia, trained as he must have been at a school of medicine of some University, could possibly display such gross ignorance of the practical details of aseptic operative surgery as the author throughout this work assumes. Far too much space is occupied in describing the elementary minutiae of the requisite preparation for operation, and much of it might well have been left to the practitioner's resourcefulness and ingenuity. The book is stated, in the Preface, to have been started as a guide to the general practitioner in the country who "thinks that he would like to try and remove a diseased appendix". If his ignorance be anything approaching what the author would have us believe, it would, we consider, be very much better to discourage rather than encourage him to undertake "His First Appendix Operation", which was the title originally intended for this work.

The author has, himself, evidently had a ripe experience in the diagnosis and treatment of appendicitis—such an experience as commands respect for his opinions, although some of these are distinctly unorthodox. He believes that "chill is the chief cause of appendicitis in those persons who are predisposed to catarrh by the inheritance of exuberant lymphoid tissue in the appendix, which is almost invariably accompanied by an exuberance of lymphoid tissue in Waldeyer's circle in the nasopharynx", and that "children and adults who have enlarged tonsils always have a large appendix: people who have a large appendix are often subject to catarrh of the colon and appendix".

In the clinical investigation of a case of acute appendicitis the importance of vaginal examination in a married woman is very rightly stressed, but no mention is made of the equally important rectal examination in males and young females. In describing rigidity of the abdominal wall no proper distinction is drawn between 'voluntary' and true 'involuntary' rigidity, nor is their differential diagnosis discussed. On the whole the technical details of the operative procedures recommended for the different types and stages of acute appendicitis are admirably described and are such as we can commend—with a few exceptions. Thus, in operating for appendicitis with diffuse peritonitis it is advised (p. 201) that after removal of the appendix a fresh opening should be made in the median line and the peritoneal cavity well flushed out with warm saline solution—a practice we had thought quite obsolete; while in opening an appendicular abscess transperitoneally the protection of the peritoneal cavity by gauze packing—a very important matter—is very inadequately described. The advice given as to the non-removal of the appendix in large abscess cases is extremely sound, all the more so when proffered to one undertaking "his first appendix operation". Several good recipes and some useful hints on diet are given in an appendix.

The Thyroid: Surgery, Syndromes, Treatment. By E. P. SLOAN, M.D. Edited by Members of the Sloan Clinic. With a Foreword by WM. SEAMAN BAINBRIDGE, M.D. 10½ × 7½. Pp. 475 + ix, with 99 illustrations. 1936. London: Baillière, Tindall & Cox. 45s.

THIS volume makes no pretence of being an encyclopædic work of reference, but embodies the working theories and conclusions, unhappily posthumous, of a surgeon who devoted twenty-five years of his life exclusively to goitre work. That his practical experience was abundant is evident from the statement that at the time of publication some 15,000 cases of goitre had been operated on in the Sloan Clinic. There is, indeed, evidence in the book that the vast amount of routine operative work has militated against scientific investigation to some extent, for the sections dealing with the details of operative technique and surgical anatomy are more convincing than those concerned with etiology and pathology. The author elaborates a theory that there is in secondary toxic goitre an 'anaphylactic group' of cases in which such symptoms as urticaria and asthma are due to absorption of certain unspecified degeneration products. No attempt, however, appears to have been made to establish this interesting theory upon a sound experimental basis. The theory that toxic goitre is a dys-thyroidism rather than a hyperthyroidism is supported, and attention is concentrated on the thyroid gland, with little consideration of the part played by the hypothalamus and the pituitary in initiating the hyperplastic change in the thyroid. There is no mention of Harington's important researches on thyroxine. The proof-reading leaves much to be desired, and it is irritating to find 'septæ' recurring as the plural of septum, and to be told that "the thymus measures 5 c.c. in length and 4 c.c. in width".

Die Nachbehandlung nach Operationen: ein Lehrbuch in Vorlesungen. By Prof. Dr. PAUL REICHEL, Geheimer Sanitätsrat. Third edition. Large 8vo. Pp. 499 + xii, with 85 illustrations. 1936. Munich: J. F. Bergmann. Paper covers, RM. 24; bound, RM. 25.80.

THIS book, which is in the form of forty-two lectures, is of especial value in describing and discussing the after-treatment of operation cases because it represents the matured experience of its distinguished author. The first edition was written in Breslau in 1896, when he had only ten years' experience as an assistant but was filled with the enthusiasm of youth. The second was from Chemnitz in 1908, and the present, in 1936, when he has had more than thirty years' experience as the director of a large hospital in Munich.

The work is divided into two parts: the first, of fourteen lectures, deals with general surgical problems, concluding with a discussion of the role of physiotherapy, in which mechanical apparatus plays a larger part than we are accustomed to assign to it in this country.

The second part, consisting of twenty-eight lectures, deals with all the organs of the body, and special surgical departments. Of these no less than eight are concerned with laparotomy and operations on the stomach and intestines. The last eight lectures deal with the after-treatment of injuries and operations on the limbs. A specially useful feature of this section is the discussion about prognosis and insurance liabilities.

Chirurgische Operationslehre. By Dr. MAX SAEGBESSAR, Privatdozent an der Universität Bern. Crown 8vo. Pp. 189 + vi, with 132 illustrations. 1935. Berlin: Julius Springer. Paper covers, RM. 7.50; bound, 8.60.

THIS short and compact book is primarily a guide to an operation course on the cadaver. As it represents the teaching of the famous Bern School, and is introduced by De Quervain, its authority is beyond question. Its main value is the many anatomical diagrams, which are models of accuracy and clearness. As in all books of this kind, the space devoted to the ligature of arteries and amputations is out of all proportion to their importance in clinical surgery.

Recent Advances in Genito-urinary Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital; Surgeon and Urologist, Essex County Council; and NORMAN M. MATHESON, M.B., F.R.C.S., M.R.C.P., Surgeon, Central Middlesex County Hospital. Large post 8vo. Pp. 213 + viii, with 89 illustrations. 1936. London: J. & A. Churchill Ltd. 15s. net.

THIS is one of the latest additions to the "Recent Advances" series, and it is no mean praise to state that it keeps up to the standard of the previous volumes. It is well written and attractively published; in it the authors more than justify their attempt to place before their readers recent innovations as they affect the commoner conditions encountered in urological practice.

Amongst the many excellent discussions we should select that on the etiology of urinary stone as one of the best; it embodies all the most recent work on the subject and lays emphasis on the undoubted relation between calculus formation and endocrine dysfunction; since renal colic and hæmaturia may antedate the clinical manifestations of hyperparathyroidism, it seems probable that some, at any rate, of those disheartening cases of recurrence of stone in the kidney may owe their cause to this disease.

On two points we venture to suggest that the authors do not express the current opinion of urologists; they do not mention the median subumbilical incision for the removal of stone from the pelvic ureter—a surprising omission—and they assert that most surgeons now remove the ureter "in its entirety" when excising a tuberculous kidney; we own that they add that this technique is not universally agreed on, but, if it be done with the object of avoiding a subsequent sinus in the loin, we believe that it is unnecessary.

There are excellent sections on pyelography, on renal function tests, on the role of sympathectomy in urology, and on modern methods of treating the enlarged prostate.

The numerous illustrations are hardly up to the high standard to which Mr. Hamilton Bailey has accustomed us, but this may be explained by the fact that they have been borrowed. The bibliography at the end of each section is admirable, and is exactly what is needed by the busy surgeon.

This handy little book contains a vast amount of useful and interesting information, and we have no hesitation in recommending it to the readers of this JOURNAL.

Surgical Diseases and Injuries of the Genito-Urinary Organs. By Sir JOHN THOMSON-WALKER, D.L., M.B., C.M.Ed., F.R.C.S.Eng., Consulting Urologist and Emeritus Lecturer in Urology, King's College Hospital, etc. Second edition, revised: edited by KENNETH WALKER, M.A., M.B., B.C.(Cantab.), F.R.C.S..Eng., Jacksonian Prizeman and Hunterian Professor, R.C.S., 1911, 1922, 1924, 1933; Lecturer in Venereal Diseases, St. Bartholomew's Hospital; etc. Medium 8vo. Pp. 974 + xviii, with 283 illustrations and 25 colour and 33 black-and-white plates. 1936. Cassell & Co. Ltd. 32s. 6d. net.

In a review of the first edition of this book, in 1914, we ventured to predict that it would quickly take its place as a standard text-book in urology; this has been verified, and the work has proved of the greatest service to the general surgeon as well as to the specialist.

This new volume is a credit to the authors, the publishers, and the artists concerned; it is well bound and clearly printed, and the illustrations are not only well reproduced and of artistic merit, but they are of real help in elucidating the text.

Owing to the advances in genito-urinary surgery during the last twenty years it has been found necessary to write new chapters on renal function tests, on transurethral operations,

on obstruction at the bladder neck, and on impotence and sterility; these have been admirably done, and give a clear and up-to-date account of our present knowledge of these subjects. For the same reason those sections which treat of prostatic enlargement, of renal tuberculosis, and of bladder tumours have had to be rewritten; the subject of the use of X rays in diagnosis and treatment has been dealt with more fully, and the recent developments in pyelography have necessitated many additions to both the text and the illustrations.

We cannot help thinking that the numerous readers of this rather monumental text-book will feel a sense of disappointment, mingled with a mild surprise, when they eagerly turn to certain parts to find out what two such well-known urologists have to say on some of the very latest advances in treatment.

For instance, we not unnaturally looked up the treatment of the *Bacillus coli* infections by the use of mandelic acid; we own to feeling slightly shocked when we found no mention of this drug either in the text or the index. In the same way, we sought in vain for any reference to the treatment of retention of the testis by endocrine therapy.

It may quite well be that the authors consider that the introduction of mandelic acid is too recent for its value to be properly assessed, and that they disapprove of the use of injections which favour the descent of the testis, possibly by bringing about a premature development of the secondary male characters. We think, however, that the student of surgery would expect to find in a modern volume of urology a discussion on such important points and some guidance as to their value; moreover, at the higher examinations in surgery, a candidate would surely be required to know something on such subjects.

We hesitate to criticize the chapter on tumours of the suprarenal gland, for it might be urged that this organ belongs neither to the urinary nor to the genital apparatus; since, however, several pages have been devoted to these neoplasms we record our surprise that it has not been brought up to date. The astounding changes that may occur in the female afflicted by such a growth—the moustache and beard, the apical baldness, the general hirsuties, the amenorrhœa, the rise of blood-pressure, and the alteration in the sexual outlook—are not mentioned; no reference is made to the late Sir Percy Sargent's successful case of operation, nor to any recent literature on the subject, though several cases have been published in the last few years. Indeed, with the exception of a reference to an article by Broster in 1931, the bibliography at the end of this chapter is quite out of date.

An excellent chapter on Stone in the Ureter is similarly marred by what we think must have been an oversight of the authors. In discussing the operative treatment of stone in the pelvic ureter they state that "the median subumbilical incision and extraperitoneal approach is far superior to the lateral curved incision": a remark with which every surgeon who has tried the former operation will most emphatically agree. It is disconcerting, therefore, to find under the heading of operations on the ureter, a long description of what is admittedly the inferior technique and a quite inadequate account of that previously recommended; it is, in fact, dismissed in some seven lines, which would be of very slender assistance to any surgeon who was contemplating carrying out such an operation.

Apart from these minor defects, which we recognize as liable to be included in any work of this magnitude, we have nothing but praise for the book and we cordially commend it to our readers.

In the inevitable future third edition we hope the authors and publishers will have mercy on the poor reader and issue the work in two, or even three, volumes; this *opus ponderosum* weighs just over five pounds, and it is impossible to read it in comfort.

Surgery for Dental Students. By PHILIP H. MITCHNER, M.D., F.R.C.S., C. E. SHATTOCK, M.D., F.R.C.S., E. G. SLESINGER, B.Sc., F.R.C.S., and CECIL P. G. WARELEY, D.Sc., F.R.C.S. 9 × 6. Pp. 364 + ix, with 105 illustrations. 1936. London: Baillière, Tindall & Cox. 12s. 6d.

THE authors aim particularly at covering the Syllabuses in Surgery and Surgical Pathology of the Royal Colleges of England, Scotland, and Ireland, and of the British Empire Universities: they do more, as they have succeeded in producing a concise and practical text-book for dental students.

The general surgical field is touched with a light hand, yet with sufficient detail to give the student a working insight into underlying pathological and clinical processes; coming

to the territories where a working knowledge of surgery is of importance the teaching becomes fuller and more detailed, and is of great practical value in dealing with the problems common to the surgeon and dentist.

The skill with which condensation has been carried out without loss of efficiency should make the book popular with students and a convenient manual for reference after qualifying.

Diagnostik mit freiem Auge Ektoskopie und Ektographie mit einem Anhang neuere Tast-befunde. By Dr. EDUARD WEISZ and Dr. L. SCHMIDT. Fourth edition. Royal 8vo. Pp. 176 + viii, with 84 illustrations. 1933. Berlin and Vienna: Urban & Schwarzenberg. Paper covers. RM. 9.50; bound, RM. 11.

THIS monograph is a description of how much can be learned by mere inspection with the naked eye and by palpation. In the thorax the movements of the intercostal muscles, and in the abdomen the movements of the abdominal muscles, on respiration and on speaking are described in great detail, and especially as these movements are affected by various morbid conditions. In the present day, when so much reliance is placed upon diagnosis by laboratory and instrumental methods, it is well to be reminded of how much can be learned by the trained eye and finger.

Gestes et Procédés techniques de Chirurgie Générale. By JEAN BERGER, Chirurgien des hôpitaux de Paris. 9½ + 6½ in. Pp. 138, with 124 illustrations. 1936. Paris: Masson et Cie. Fr. 32.

THIS is a refreshingly original book. It is not a text-book of operative surgery, but a manual dealing with surgical technique. It describes not so much what to do as how to do it. It shows how to hold a pair of scissors and to cut a ligature; how to handle a needle, and to put on a pair of forceps; and a hundred other points of technique. It is good, and will repay study by any surgeon who thinks about, and wishes to polish, his methods.

A Descriptive Atlas of Radiographs. An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Third Edition, revised and enlarged. Cr. 4to. Pp. 560 + xxxi, with frontispiece and 794 illustrations. 1936. London: Henry Kimpton. 42s.

THE fact that a third edition of this book has been called for in ten years shows that its value has been appreciated.

It consists of 794 cases in which an X-ray print is accompanied by a short description and sometimes a clinical history. In the present edition two new sections appear, one on medico-legal problems, chiefly the determination of the age of the fœtus, and one on anthropology, which contains only two figures, one of which is very obscure. In his introductory remarks the author makes a well-argued appeal for a museum of radiography. As a book of reference it is invaluable, but its usefulness will be improved as time goes on by the inclusion of more radiographs of difficult cases, whilst the obvious things might be omitted. For example, in the section on fractures, there is only one figure of a fractured os calcis, whilst many of the common fractures of the shafts of the long bones hardly need the wealth of illustration given to them.

Clinical Handbook for Residents, Nurses, and Students. Being the Routine Methods of St. Vincent's Hospital, Sydney. By Members of the Staff of the Hospital. Edited by VICTOR M. COPPLESON, Ch.M., F.R.C.S., F.R.A.C.S., and DOUGLAS MILLER, Ch.M., F.R.C.S., F.R.A.C.S. Cr. 8vo. Pp. 205 + x. 1936. London and Sydney: Angus & Robertson Ltd. 6s.

It is obvious that a manual of this sort compiled by the members of a hospital staff for the guidance and direction of their own students, nurses, and residents, must be of great practical value.

The chapters on clinical pathology and biochemistry, giving as they do an explanation and description of all ordinary methods, appear to us to be of the greatest value. Also the

many reference tables, e.g., those of the normal content of various substances such as urea, calcium, etc., in the blood, are most useful. It is curious that whilst the management and nursing of ophthalmic, E.N.T., skin, and gynaecological cases is described, there is no section on the management and nursing of general surgical or fracture cases, nor of the technique of radium application. The opening section on sterilization is excellent, but we think it illogical to describe a special '4-day' method for operations involving bones and joints, which implies that the method used for other cases is inadequate. The method of cutting off plaster casts with a knife seems unnecessarily risky.

Baillière's Synthetic Anatomy. By J. E. CHEESMAN. Part XIII. The Eye and Orbit. Plates $7\frac{1}{2} \times 9$ in. 1936. London: Baillière, Tindall & Cox. 3s. net. Complete set of 14 parts in Binder, 45s. net. Loose Leaf binding case, to take whole work, 3s. net.

WE have already given an appreciative review of the early parts of this work, in which we described it as "the best and most convenient substitute for the body". The last section, on the orbit, consisting of eight coloured plates, completes the series. It consists of superimposed coloured pictures on a transparent medium, which allows each layer to be seen through the one above it.

The facts that the work has been produced in French, German, and Spanish, and that over 100,000 copies have been issued, are sufficient evidence of its proved value in the teaching of anatomy.

Text-book of Pathology. By Sir ROBERT MUIR, M.A., M.D., Sc.D., LL.D., F.R.S., Professor of Pathology, University of Glasgow; Pathologist to the Western Infirmary, Glasgow. Fourth edition. Medium 8vo. Pp. 994 + viii, with 571 illustrations. 1936. London: Edward Arnold & Co. 35s. net.

THIS is a new edition of the well-known text-book on pathology, and no further notice seems to be required than the statement that the incorporation of important additions to knowledge that have occurred since the last edition ensures that the book remains the standard work in pathology in this country.

Die Wirbelgelenke. By Prof. Dr. MAX LANGE, Director of the Orthopædic Clinic, Munich. Second enlarged edition. Large 8vo. Pp. 138 + viii, with 94 illustrations. 1936. Stuttgart: Ferdinand Enke. RM. 10.

THIS is a very careful study of the anatomy, physiology, and pathology of the intervertebral joints. The difficulties of accurate X-ray examination are stressed, and clear directions given for overcoming these. Then in great detail all conditions of altered position (e.g., scoliosis or kyphosis), and inflammatory, infectious, and traumatic conditions are described and figured from pathological specimens and radiographs. The greatest attention is paid to the changes in the joints between the articular processes rather than to those in the intervertebral discs. The book does not deal with problems of treatment.

Roentgendiagnostik der Knochen- und Gelenkkrankheiten. By Professor Dr. ROBERT KIENBÖCK (Vienna). Part 4. Degenerative Wirbelsäulenerkrankungen. Large 8vo. Pp. 229-450, with 214 illustrations. 1936. Berlin and Vienna: Urban & Schwarzenberg. RM. 24.

THIS is a very detailed account of the changes which take place in the vertebral bodies in chronic diseases of the vertebral column. It is chiefly concerned with static kyphosis, scoliosis, and spondylitis deformans. Its chief value lies in the numerous X-ray pictures of different regions of the spine and at different stages of the disease, together with careful clinical histories of the cases concerned. It forms a useful companion volume to that by Professor Lange which deals with the changes in the joints.

Die moderne Finsenbehandlung. By Dr. SVEND LOMHOLT, Director of the Dermatological Clinic at the Finsen Institute, Copenhagen. Large 8vo. Pp. 64, with 52 illustrations. 1934. Berlin and Vienna: Urban & Schwarzenberg.

THIS forms but a chapter in a larger work on treatment by radiotherapy. It gives in simple language the principles underlying the therapeutic use of light, both sunlight and that of various carbon and ultra-violet lamps.

The modern Finsen lamp is a carbon-arc lamp with special lenses and filters, and it is claimed to be more efficient than the older pattern. Thirty advanced cases of lupus vulgaris are presented at different stages of treatment, showing the progress towards recovery made under the Finsen light treatment.

Behandlung der Verletzungen und Eiterungen an Fingern und Hand. By Professor Dr. M. ZUR VERTH, Privatdozent in the University of Hamburg. Second edition. 5½ × 8½. Pp. 164 + viii, with 59 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 8.70; bound, RM. 9.60.

TRAUMATIC surgery has assumed a much more specialized aspect in Germany than in this country. This is partly due to the system of state insurance of workmen which has made available a great mass of statistics showing the relative economic importance of different forms of injury. Over 34 per cent of all finger injuries lead to some degree of permanent disability. The amounts paid in compensation for cases of finger injuries is almost double that paid for all cases of fracture of the long bones. This little book is a clearly written and well-illustrated account of all the different types of injury and infection to which the hand and fingers are subject, and the most effective way of treating the same. It concludes with a useful discussion of the different types of amputation of the fingers and hand suitable for hand workers and brain workers.

Die Kurzwellenbehandlung in der Chirurgie. By Dr. ALFONS LOB, Roentgenabteilung der Chirurgie, Univ.-Klinik, Munich. Medium 8vo. Pp. 68. 1936. Stuttgart: Ferdinand Enke. RM. 3.60.

THIS is a short and simple explanation of the physical nature and action of short-wave therapy. The author holds that the early and exaggerated promises of the therapeutic value of this method have not been fulfilled. Especially is this the case in the treatment of acute pyogenic affections, in which short-wave therapy can do no more than act as an adjuvant to the ordinary surgical treatment. With new growths, too, the short-wave therapy has given doubtful benefit, and that chiefly when combined with X-ray treatment. The principal value of the method according to the Munich experience is in the treatment of sub-acute and chronic affections.

The Patient Looks at the Hospital. By FLORENCE G. FIDLER. With a Foreword by JOAN MALLESon, M.B., B.S. Crown 8vo. Pp. 128 + xii. 1936. London: Robert Hale & Co. 2s. 6d. net.

THIS is a useless book which illustrates the fallacy of arguing from a particular to the general. Miss Fidler appears to have had some unfortunate experience in a voluntary hospital. She deduces therefrom that matrons are martinets, nurses are fools, doctors are hoodwinked, and committees' interest lies more in the collection of money than in the welfare of the hospital inmates. She views everything with a jaundiced eye and seems to be wholly ignorant of life in a well-organized hospital. The book is dedicated "to the Subscribers to all Voluntary hospitals". Dr. Joan Malleson writes a discrete foreword.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

- St. Bartholomew's Hospital Reports.** Edited by W. G. BALL, G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, H. H. WOOLLARD, R. C. ELSLIE, J. P. ROSS, and J. MAXWELL. Vol. LXIX. Demy 8vo. Pp. 413 — xxvi. Illustrated. 1936. London: John Murray. 21s. net.
- Illustrations of Regional Anatomy.** By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. Fcap 4to Section VI, Upper Limb, 42 Plates, 7s. 6d. Section VII. Lower Limb, 52 Plates, 10s. 1936. Edinburgh: E. & S. Livingstone.
- Disability Evaluation: Principles of Treatment of Compensable Injuries.** By EARL D. MCBRIDE, B.S., M.D., F.A.C.S., Assistant Professor in Orthopedic Surgery, University of Oklahoma. Large 8vo. Pp. 623 — xvi, with 374 illustrations 1936. London and Philadelphia: J. B. Lippincott Co. 38s. net.
- Textbook of General Surgery.** By WARREN H. COLE, M.D., F.A.C.S., Professor of Surgery, University of Illinois College of Medicine, and ROBERT ELMAN, M.D., Associate Professor of Surgery, Washington University School of Medicine, St. Louis. Large 8vo. Pp. 1031 + xvi, with 559 illustrations. 1936. London: D. Appleton-Century Co Inc. 40s. net.
- Modern Urology in Original Contributions by American Authors.** Edited by HUGH CABOT, M.D., LL.D., C.M.G., F.A.C.S., Professor of Surgery, The Mayo Foundation, Graduate School of the University of Minnesota; etc. Third edition, thoroughly revised. Royal 8vo. In two volumes. Vol. I, pp. 951, with 546 illustrations and 12 plates; Vol. II, pp. 862, with 374 illustrations and 9 plates 1936. London Henry Kimpton. 90s. net.
- Essentials of Oral Surgery.** By Professor V. P. BLAIR, A.M., M.D., F.A.C.S., Washington University, St. Louis, and Professor R. H. IVY, M.D., D.D.S., F.A.C.S., University of Pennsylvania, Philadelphia, with the collaboration of Associate Professor J. B. BROWN, M.D., F.A.C.S., Washington University, St. Louis. Second edition 9½ — 6 in. Pp. 606, with 445 illustrations. 1936. London: Henry Kimpton. 30s. net.
- The Digestive Tract: A Radiological Study of its Anatomy, Physiology, and Pathology.** By ALFRED E. BARCLAY, O.B.E., M.A., M.D., D.M.R. & E., Honorary Radiologist to the Nuffield Institute for Medical Research, Oxford. Second edition 7½ — 9½ in. Pp. 427 — xxxvi, with 296 illustrations, including 23 Plates 1936. London: Cambridge University Press. 36s. net.
- Urological Roentgenology.** By MILEY B. WESSON, M.D., Ex-President, American Urological Association, and HOWARD E. RUGGLES, M.D., Roentgenologist to University of California Hospital, St. Luke's Hospital, and Clinical Professor of Roentgenology, University of California Medical School. 6½ — 9½ in. Pp. 269, with 227 illustrations. 1936. London: Henry Kimpton. 22s. 6d. net.
- Medizinische Praxis. Band XXI. Anleitung zur Schmerzbetäubung.** By Prof. Dr. FRITZ F. HÄRTEL, Direktor der chirurgischen Abteilung des Oskar Ziethen-Krankenhauses Berlin-Lichtenberg. Large 8vo. Pp. 106 — x, with 17 illustrations. 1936. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 10, bound. RM. 11.50.

Pathologie der Funktionen und Regulationen. By Dr. L. LICHTWITZ, Head of the Medical Department of the Montefiore Hospital, New York; Clinical Professor of Medicine, Columbia University. Large royal 8vo. Pp. 332 + viii, with 77 illustrations, mostly plates, and 61 graphs. Leiden: A. W. Sijthoff's Uitgeversmaatschappij N.V. Paper covers, Fl. 13.25; Bound, Fl. 14.50.

The Operations of Surgery. By R. P. ROWLANDS, M.S. (Lond.), F.R.C.S. (Eng.), Late Surgeon to Guy's Hospital, etc.; and PHILIP TURNER, B.Sc., M.S. (Lond.), F.R.C.S. (Eng.), Consulting Surgeon to Guy's Hospital, etc. Eighth edition. Vol. II: The Abdomen. Royal 8vo. Pp. 998 + ix, with 514 illustrations (4 in colour). 1937. London: J. & A. Churchill Ltd. 36s. net.

Tuberculosis, Cancer and Zinc. An Hypothesis. By DOUGLAS BARRON CRUICKSHANK, L.R.C.P. and S., L.D.S. (Edin.), D.P.H. (Camb.), Member of Research Staff, Papworth Village Settlement. With an Introduction by SIR PENDRILL VARRIER-JONES, M.A. (Cantab.), F.R.C.P. (Lond). Demy 8vo. Pp. 75 + xvi. 1936. London: Medical Publications Ltd. 7s. 6d.

Kidney Pain: Its Causation and Treatment. By J. LEON JONA, D.Sc., M.D., M.S., F.R.A.C.S., Hon. Assistant Gynæcological Surgeon, Women's Hospital, Melbourne. Large post 8vo. Pp. 94 + viii, with 61 illustrations. 1937. London: J. & A. Churchill Ltd. 7s. 6d.

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, K.B.E., LONDON

XIII. SIR WILLIAM STOKES, 1839-1900 : AN EARLY ADVOCATE OF LISTERIAN SURGERY

SIR WILLIAM STOKES came of an Irish family distinguished for its intellectual ability through several generations. He was the second son of Dr. William Stokes, F.R.S., Regius Professor of Physic in the University of Dublin, a man of world-wide renown both as a physician and a scholar. His name remains in 'Cheyne-Stokes respiration'. Sir William Stokes's elder brother was Whitley Stokes, C.S.I., C.I.E., a Celtic scholar and a member of the Legislative Council of India.

Educated at the Royal School, Armagh, William Stokes entered Trinity College, Dublin, graduated in Arts at the age of twenty, and proceeded to the M.D. in 1863. He then spent two years on the Continent and became fluent in French and German. He was elected Surgeon to the Meath Hospital in 1864, transferred to the Richmond Hospital in 1868 and returned to the Meath in 1888. He was elected Professor of Surgery in the Medical School attached to the Royal College of Surgeons of Ireland in 1872, became a Fellow of the College in the following year and served as President in 1886. He received the honour of Knighthood at the hands of Lord Aberdeen, then the Lord-Lieutenant, in June, 1886, and in 1892 was appointed Surgeon-in-Ordinary to the Queen in Ireland. During the South African War he offered his services, and in January, 1900, went out as a consulting surgeon to the British Expeditionary Force. He entered Ladysmith soon after its relief and did much good work in various hospitals. He overtaxed his powers, however and died of pneumonia, complicated by chronic nephritis, at Pietermaritzburg, Natal, on August 18, 1900.

A highly cultivated gentleman, Stokes had the gifts of oratory and of humour. The passages quoted below show, too, that he was a master of English. They are taken from the address which he delivered at the Worcester meeting of the British Medical Association in 1882. Their interest lies in the fact that Listerian surgery still needed the active support of British surgeons :—

"Among many depreciatory remarks that have been made in reference to Listerism is one based on its alleged want of originality. It has been stated that

both antiseptic principles and practice were understood, recognized, and appreciated by many of Mr. Lister's predecessors and contemporaries. Foremost among the latter, M. Maisonneuve has been mentioned. Having attended the clinique of that eminent surgeon for two sessions in 1864-5, I am in a position to mention the nature of the wound-dressings then employed by him. With a large syringe, a quantity of a weak solution of *acide phénique* was applied to the wound, then a piece of linen or cloth, perforated with numerous openings and covered with a yellow-coloured grease, was placed on the wound, secured by a dry compress and



SIR WILLIAM STOKES

bandage. Such were the antiseptic dressings of which Lister's, it is alleged, are only a somewhat complicated, expensive, and in many cases dangerous reproduction!

"Much blame has been cast on Professor Lister and his followers for not having had recourse more largely than they have done to statistics to prove the superiority of antiseptic practice over the older and alleged simpler methods of wound-dressing and to show that by the use of the former we are more independent of those epidemic influences that have hitherto been so pregnant with disaster in operative surgery. It is not my purpose here to discuss the value of the surgical statistics that have been adduced to prove that the alleged simpler methods of wound-dressing are of equal efficacy to those of Lister, especially as most of them have a strange family resemblance to the latter; but this I will say, that, whatever

value is to be ascribed to accumulated figures—often sadly fallacious—that value is not to my mind greater, or at all so great, as the often-repeated occurrence of test cases recorded daily, not alone in a particular hospital, town, or country but in hospitals in all climates and conditions, where the hygienic surroundings are brought to the highest known degree of perfection, as well as where they are in a condition the most deplorable. Such records carry more weight with me than the inflated statistics from any particular hospital, or the alleged results obtained without antiseptics after any special operation or group of similar operations.

“It is a subject of regret to me that so many surgeons have been found of long experience and of great and deserved eminence, who have either been disposed to discredit a thorough antiseptic practice altogether or have given but a very lukewarm adherence to it. Much allowance, however, must be made for the well-known and not unnatural dislike to change on the part of those, many perhaps advanced in life, whose early training has been so different to that now available. With their successors, more fortunately circumstanced in this respect, the case is different. Their condemnation has, I fear, been the result of apathy, indifference and in some instances indolence, preventing them taking the trouble to learn either the principles or the details of the practice.

“These representatives of what may be termed a Rip Van Winkle school of surgery differ in one respect from the mythical personage just alluded to. His ignorance of what was going on about him was the result of involuntary unconsciousness. But his surgical analogues, I fear, wilfully refuse to see, and wilfully refuse to acknowledge what has been and is being done. Strangely unmindful of the fact that honest scientific toil has never yet proved other than fruitful of the good, they promulgate views ever acceptable to ignorance and indolence, and make the land ring with the false and cruel tale that the value of Listerism is a delusion, a bubble, a shadow and a myth, at once expensive, complicated and poisonous. If, on this last account, it is to be rejected then may we, with equal justice, say: ‘Away with anæsthetics—away with opium, mercury, belladonna—with half, or more than half the means at our disposal for alleviating human suffering and prolonging life.’

“In the interests, and for the credit, of British surgery, it is time so unrighteous a warfare should cease. It is time that the irritating dust of an unreasoning prejudice should be contemptuously swept away. It is time that one of the greatest discoveries and boons to surgery this country has produced should be universally recognized as such. It is time that its discoverer and exponent should be acknowledged as one of whom it may be well said ;

‘With Genius Nature joins, in everlasting covenant still ;
The promises of one, the other fails not to fulfil.’”

The portrait of Sir William Stokes in his robes as President of the Royal College of Surgeons of Ireland is copied by kind permission of Messrs. Baillière, Tindall & Cox, from the frontispiece to Dr. William Taylor’s edition of the *Selected Papers on Operative and Clinical Surgery of William Stokes*.

JOHN HUNTER IN THE CAMPAIGN IN PORTUGAL, 1762-30*

BY G. E. GASK

IN 1934 there came into the market a bundle of manuscripts which had been in the possession of John Campbell, Fourth Earl of Loudoun (1705-83), who commanded the British Forces in the Portuguese Campaign of 1762-3. The great interest of these papers lies in the fact that John Hunter served as a military surgeon in this war. The attention of Sir D'Arcy Power, Honorary Librarian of the Royal College of Surgeons, was called to the papers, and he, quickly realizing their value and special significance, advised the College to buy them.

There were over 300 documents in the bundle, all dealing with the medical arrangements of the campaign. In those days the Commander-in-Chief dealt directly with his medical officers, it being only later, when the conditions of war became too complicated and onerous for one man to direct, that the appointment of a Director-General of Medical Services became imperative. In consequence we find that these documents were directed to the Earl of Loudoun, who dealt with them, endorsed them with his own hand apparently, and preserved them. They include a large number of returns of sick and convalescents, hospital states, official accounts, letters from medical officers asking for leave, complaints, requests, and among them a number of letters from Hunter himself.

These original documents are of great interest to us, for they reveal a chapter, previously unknown, in the life history of John Hunter. In addition, however, they throw light on the state of the medical services of that day. By means of this new evidence we learn with accuracy the composition of the Hospital of the Expeditionary Force, and we are able to recapture something of the mode of life and trials and tribulations of the members of its staff, and to reconstitute the intimate details of army life.

It should be remembered that in the eighteenth century military hospitals were still in their infancy. The national conscience that soldiers were not merely food for cannon, but that they were husbands, brothers, and sons to be saved and cared for was only slowly awakening. The account to be given below will reveal that in this campaign great efforts were made to establish a first-rate hospital according to current opinion, well equipped with medical staff, with medicines, with instruments, bedding utensils, necessities of all kinds, and with women nurses.

POLITICAL REASONS FOR THE CAMPAIGN

In order to give some idea of the scene in which Hunter played his part, it is necessary to glance briefly at the political reasons that led up to the campaign.

England was embroiled in the Seven Years War (1756-63), the ramifications of which are hard to follow. Frederick the Great of Prussia was fighting for

* From the Loudoun Letters in the Library of the Royal College of Surgeons of England.

Silesia, and had against him the French, Austrians, Russians, and Swedes. Britain made an alliance with Frederick and sent men and money to help him, less it may be imagined because of our love for Frederick than on account of our fear of France. While British troops were fighting in Europe, active operations most eventful for us were occurring in India and America. The French were turned out of India, and America was won for the Crown. Fortescue actually held that Pitt's insight turned the whole war in Europe into a diversion in England's favour. It must also be remembered that in England there was always the dread of a French invasion. A French expedition was planned for a descent on England, and eighteen thousand men lay ready to embark on board the French fleet when Hawke found and smashed it at Quiberon Bay.

This ever-present fear of invasion meant that the British fleet had to keep an incessant watch and blockade of the French coast. They needed badly a port to put into from time to time to refit and victual, some place not so far away as the home ports; or, in the words of Fortescue, "a place of refreshment." This accounts for the attack on Belleisle, when our John Hunter comes into the story. Belleisle was taken in June, 1761, but whether it was much use to us when we had got it remains obscure.

Fighting still went on in Europe, and now Spain joined in the fray and we declared war against her.

In 1762 Spain, on the pretext of Portuguese friendship with England, invaded Portugal and overran the country as far as the Douro (Fortescue, *History of the British Army*, ii, 554). The Portuguese applied to England for aid, and as they were, and still remain, our Ancient and Honourable Allies, they did not ask in vain. An expeditionary force was got together and sent to help them. Orders were sent to Belleisle for the despatch of four regiments of infantry, the 3rd (The Buffs), the 67th (Boscawen's), the 75th (Crawford's), and the 85th, together with detachments of the 16th Light Dragoons. Two more regiments were added from Ireland—Armstrong's and Blayney's—bringing the total up to about seven thousand men. The two regiments from Ireland came into the Tagus by the first week in May, 1762, but it was not until June that the rest of the troops arrived with the Count of Lippe Bückeburg, the famous artilleryist, as Commander-in-Chief of the Allied Forces, and Lord Loudoun in command of the British (Fortescue).

This Portuguese campaign was really a sideshow, and a very unimportant one at that, considering that fighting extended over a large portion of the civilized world, and it does not appear to have influenced in any degree the final peace. The operations were so trifling that Fortescue deemed them unworthy of detailed mention. The Spaniards captured Almeida early in August, 1762, and Count Lippe was obliged to remain on the defensive and cover Lisbon at the line of the Tagus. We shall see later that the hospitals with which Hunter was concerned were situated all along this river. There was hardly any fighting and there were very few wounded, and this, from a military point of view, quite unimportant campaign came to an end with the Peace of Paris on Feb. 10, 1763. By this treaty France ceded to England the whole of India except Pondicherry and Chandernagor, and also the whole of Canada, and Spain gave up Florida.

After this brief introduction to the history of the war we may turn to see how Hunter came to be involved, and the share he took in it.

At the age of 20 Hunter came to London following a letter to his successful brother, William, in which he offered to act as William's assistant in his anatomical work, or, if that proposal should not be accepted, expressing a desire to go into the Army (Home, quoted by Peachey, *A Memoir of William and John Hunter*).

As is well known, for the next ten years Hunter worked hard in London, and then, says Ottley, "His health was beginning to suffer from his incessant studies. In the spring of 1759 he was attacked with inflammation of the lungs, which left behind symptoms that threatened to end in consumption. He was strongly advised therefore to leave London for a time, and seek a more southerly climate. With this view he applied for an appointment in the army, and was immediately made Staff Surgeon by Adair, who was then Inspector-General of Hospitals."

Whether failing health was the real reason for Hunter joining the army we shall probably never know. For those who knew the conditions of the Great War, service abroad does not sound exactly like a rest cure. We have to remember, though, that ten years previously Hunter had expressed a desire to go into the army, and it seems quite likely that to a man of spirit, and Hunter had plenty of spirit, the stirring times in which he lived made a call that he could not resist.

Whatever was the true reason which sent him to the Wars the fact remains that on Oct. 30, 1760, he was given a commission by Robert Adair, the Robin Adair of the well-known ballad. Five months later, on March 29, 1761, Hunter sailed from Spithead with an expeditionary force of ten thousand men under General Hodgson to capture Belleisle. The story of the siege and capture of this place has already been told in the many accounts of the life of Hunter, and the letters written by him from that address to William are set out in full by Peachey. It is related that Hunter did not seem best pleased with his life at Belleisle, for on July 11, 1761, he writes to his brother: "We talk of going home and then to go out upon another expedition, viz., to Martinico, if there is anything in it, I would beg of you to see if I could possibly avoid it, and be put upon some land service as the Sea plays the Devil with me. I would rather stay here in this place (if a surgeon is to be left with the Troops) rather than go to the east or west Indies."

By April, 1762, it is clear that there was news of the Belleisle contingent being sent to Portugal, though Hunter is not certain whether he will go with it. He writes thus on the 12th of that month: "Dear Brother, . . . There is nothing talked of here but Portugal. Mr. Smith our apothecary is appointed as one there; but no accounts of my going. If I am to leave this place I should like to go there but should chuse to stay here, if an Hospital was to be kept, as I suppose I shall loose my ten shilling by going (but that as it may be). . . . I hear that Dr. Blyth is to go to Portugal. I suppose Mr. Young goes as Surgeon-General, God help the Hospital when directed by such two." This is the last of the letters from Belleisle which are quoted by Peachey, whose account I have used freely.

Having now brought the reader up to date with the facts that were already known, we will proceed to examine the new evidence that has been brought to light, and set out the arrangements that were being made in London for the establishment of a hospital to serve with the Forces in Portugal.

PREPARATIONS FOR THE CAMPAIGN

The exact date on which it was decided to send an expedition to Portugal is not stated, but it is clear that by February, 1762, careful consideration was being

given to the provision of the necessary medical arrangements. Sir William Fordyce was asked to provide a "Scheme for an Hospital for 6000 men". William Fordyce at that time was Surgeon to the Third Foot Guards and had had experience of active service. Later he became Lord Rector of the Marischal College, Aberdeen (*Dictionary of National Biography*, "Munk's Roll", "Johnston's Roll"). His scheme commences :—

For 6000 men or eight Regiments and supposing each Regiment to have 100 sick. First for bedding,

One hundred Palliasses or strong Osnaburg cases to be filled with straw . . . If the Hospital is fixed and not a Field or flying Hospital there must be one hundred Bedsteads with boarded bottoms of strong work.

And he ends up by saying that a Surgeon's Chest, if fitted out, usually comes to twenty-five pounds. It is curious that no mention is made here for the care of wounded, though plenty of sick are expected. He says that unless after laying long encamped on the same ground the sick seldom exceed an hundred men in a regiment.

The Hospital System Prevailing.—At this time the medical officers to the army were divided into two classes. There were the regimental surgeons and the regimental surgeons mates who looked after the sick and wounded of their respective regiments, and then there was the Hospital which had a distinct separate medical personnel, whose duty was concerned with hospitals other than the small regimental hospitals. The Marching or Field Hospitals had disappeared after Marlborough's campaigns, and the only hospitals now were the General Hospitals. The officers of these hospitals were gathered together on the outbreak of war, as required, from the ranks of the medical profession or from those who had served in previous campaigns and were on half pay. We find that these appointments were "to our hospital", or "to the hospital", or "to the hospital appointed to attend our Forces, etc."

Lord Loudoun had already had experience of a hospital of this sort while he was Commander-in-Chief in North America. The following was its composition : "By the summer of 1757 the general hospital comprised a staff of a director and chief surgeon, James Napier, a physician Richard Huck, four surgeons, three apothecaries, twelve surgeons mates, ten apothecaries mates and a matron, Charlotte Brown, who had come over with Braddock's army" (Pargellis, *Lord Loudoun in North America*).

It was to a hospital of a similar character that Hunter was appointed as surgeon. It is probable that he wore a uniform of a staff surgeon, that is to say, a double breasted red coat with a cocked hat.

On May 4 the time of embarkment was obviously getting near, for Lord Loudoun sent a letter to John Cathcart, the Director of the Hospitals in Great Britain, saying that he had had a letter from Admiral Forbes desiring to know when all the hospital stores would be embarked. To this John Cathcart replied that the hospital stores had been put on board the *Neptune* (Captain Hubbard) and orders had been sent for it to put into Portsmouth.

On May 9 Robert Adair, Inspector-General of Hospitals, furnished Lord Loudoun with a "List of the Officers of the Hospital appointed to attend the Forces in an Expedition to Portugal", which is reproduced below in full :—

LIST OF THE OFFICERS OF THE HOSPITAL APPOINTED TO ATTEND THE FORCES
IN AN EXPEDITION TO PORTUGAL

2 Physicians	{ Dr. William Cadogan Dr. Michael Morris	
Director	William Young	
3 Surgeons	{ John Hunter William Maddox Francis Tomkins	
2 Apothecaries	{ Walter Hamilton Hugh Smith	
16 Mates	{ Jeremiah Armstrong	Regimental mate
	Croker	
	Thomas De Veil	
	David Griffiths	
	Hayes	
	Chas. Nicholas Jenty	
	{ Andrew McDowall	Old do.
	Morgan O'Brien	
	William Rodgerson	
	Anthony Robinson	
	Robert Scott	Old do.
	Edmund Tatlor	
	{ Three from Belleisle	

ROBT. ADAIR.

A subsequent document shows that the officers of the hospital were appointed as from February 10, 1762, and continued to May 14, 1763. Their rates of pay have been carefully preserved. The Director, William Young, received twenty-five shillings a day; the physicians a pound, and the surgeons ten shillings, while the apothecaries had also ten shillings and the surgeons mates only five shillings a day.

This salary was not paid weekly or monthly, nor was it paid into an agent's account in London. It was not paid until the end of the campaign, and until a certificate was produced signed by the leader of the expedition and countersigned by the Director of the hospital and by the Deputy Paymaster-General. This document is set out in full at the end of this article. All these officers, however, drew subsistence allowance in Portugal, but the amount of this allowance is not stated.

It is very interesting to note that female nurses were also included in this expedition, for a list of the Servants belonging to the hospital is given. The Matron was Mrs. Sullivan and she received half a crown a day. Then there were two head nurses, Mary Fenton and Ann Milross, and three women cooks, all at one shilling per diem. There were also five washerwomen at the same pay, and eighteen women nurses at sixpence a day each. So the washerwomen got twice as much as the nurses. (The full list is given on p. 668.)

It may come as a surprise to many that female nurses were used in military hospitals abroad before the time of Florence Nightingale. They were used, however, in the wars in Ireland in the time of William and Mary, and also in the Seven Years War in the campaigns in Germany. The presence of nurses with the forces is mentioned several times by Pringle in his book on *Diseases of the Army*, and as we have seen they were used in the war in North America in 1757.

There is no evidence as to the qualifications and training of these nurses. Pargellis thinks that they probably came from the ranks of the women who followed the army. This may be so, but the fact that there was a regular establishment with a matron and two head nurses in control makes one think that they were specially picked and appointed from home, and one would expect that they were collected from the nurses in the civil hospitals.

On May 24, 1762, Lord Loudoun addressed the following letter to William Young, the Director of the Hospital :—

I have just received information from the Admiralty that the Hospital Ships are arrived at Portsmouth and that a Ship of War is ordered to convoy them to Lisbon. It is therefore my Orders that the whole of the Hospital repair immediately to Portsmouth and there embark in order to proceed directly to Lisbon.

To this William Young replies on May 27 that everything is on board and all the commissioned officers present, and "we are to sail this afternoon if there be wind enough to take us out". So the expedition was off, and it is necessary now to see what was going on at Belleisle.

It will be remembered that four regiments of foot had been ordered to Portugal from Belleisle, and it is evident that by April they were all agog in the island, for on the 12th of that month Hunter writes to his brother, "There is nothing talked of here but Portugal". He also says that Mr. Smith the apothecary and Dr. Blyth were also going (Peachey). He was right about the apothecary, but there is no evidence that Dr. Blyth accompanied the expedition. On June 6, in another letter to his brother (Peachey), he says, "We are all busie making ready for Portugal but there seems to be no great signs".

By June 16, however, a move is imminent, for Hunter furnishes lists of the invalids of the various regiments and a return of the sick unable to embark and to be left in Belleisle. The diagnoses of the sick appear to us very loose and certainly would not satisfy the regulations for filling up the medical returns of the day, but they shed a light on the sort of material of which the British troops were made up at that time. Age and infirmities, blindness, paralysis, consumption, and rheumatism, are some of the reasons given by Hunter for incapacity to serve. Then Hunter gives a list of the officers of the hospital that are to go to Portugal. There were two officers, himself and Hugh Smith the apothecary, five mates, two servants, one storekeeper, one matron, and four nurses. Subsequent evidence shows that four mates went with him, namely, Benjamin Clark, Edward Golding, Christopher Johnston, and M. A. Clark. These one must suppose are some of "my fellow creatures of the Hospital" whom he called "a damned disagreeable lot" in his letter of September 28, 1761 (Peachey).

IN PORTUGAL

How Hunter got from Belleisle to Portugal and when he arrived there is not certain, but it seems likely that he left on the Hospital Ship *Betty* on July 6, 1762, and that he arrived in Portugal not later than July 16. This is based on the fact that Hunter made a return of sick on this ship. There were thirty-four sick, of whom nineteen were sufficiently recovered to join their regiments and fifteen had to remain. He had one case of small-pox on board.

The hospital transports, which we have seen left Portsmouth on May 27, must have arrived in Lisbon some time in June, though we have no record of the exact date.

One of the first activities of the medical service must have been to set up a hospital in Lisbon, and this appears to have been opened on June 22. It is fortunate that an excellent description of this hospital has been preserved in an official report made to the Commander-in-Chief by Colonel Cosnam, the Adjutant-General, dated August 9, 1762. One must not visualize the hospital as a neat row of army huts, with trim roads edged with festoons of whitened ropes, such as were familiar to us in many of the bases in the Great War. The general hospital in Lisbon was quartered in existing buildings, and they are thus described:—

There are four houses appointed for the service of the Hospital, viz.,

1st. The House of Lobo's (where at present the two Physicians, Master Apothecary, one Surgeon and three Mates lodge) is calculated to receive

Patients 100

2nd. The House of Panteas (?) (where one mate lodges) calculated to receive

Patients 196

N.B. There is an old Friar lodges in a little cell belonging to this house.

3rd. The Fort (where three Mates lodge) calculated to receive

Patients 150

N.B. This is much the worst Hospital, the rooms small and is removed at least a quarter of a mile from the others.

4th. Casa de Almirante (where one Surgeon and five Mates lodge) was calculated to receive

Patients 50

Provision made to receive in all

496.

Colonel Cosnam goes on to say that the staff of female nurses were attached to this hospital. From the sick return made on July 6 it appears that there were 202 patients in hospital. There would therefore have been a matron, two head nurses, and eighteen nurses to look after 202 patients. That is to say, roughly one nurse to ten patients. Colonel Cosnam evidently thought this ratio too high, for he says, "Though the hospital is at present rather overstocked with nurses for the number of sick, none can be discharged, as their places cannot be supplied on any emergency, and they expect an increase of sick daily".

This reference to the impossibility of supplying more nurses is further evidence to the opinion expressed above that these nurses were professional nurses and not recruited from the ranks of the camp followers.

Another interesting sidelight on the management of the hospital is furnished by the preservation of the Standing Orders for the discipline of the hospital given by Henry Luttrell, Deputy Adjutant-General (later second Earl of Carhampton—*D.N.B.*). The orders start modestly as if they were not accustomed to the duty: "It having been reported to General Townshend that there is not that discipline at the hospital, which can enable those that have the care of it, to do as much for the recovery of the men as he could wish. He thinks it his duty to give out the following as Standing Orders for the hospital, until he has laid them before those who will most probably give better."

A hospital guard is to be appointed and the officer of the guard is to be responsible for the regularity of the hospital and to have the rolls called before every relief so as to see if any of the patients are out. The officer is to give the doctor all the assistance in his power for such purposes as drawing water, burying the dead and in general all

kinds of labour that is above the strength of the nurses. Refractory and disobedient patients are to be confined on bread and water as long as the Doctor shall direct and a Black Hole and Irons are to be provided. The Guard will also send frequent patrols to the Tipling houses in the neighbourhood to prevent any men drinking and quarrelling with the inhabitants.

The Orders conclude with these words :—

The Officer of the Guard is to see that these orders are to be kept clean and repaired when defaced.

The paper on which these orders were written is dirty, frayed at the edges, and much worn with use, but it has survived for a hundred and seventy-five years.

By July 13 a regular return of the hospital sick on printed forms was started. From these it can be made out that between July 13 and August 6, 373 men passed through the hospital. Unfortunately only the numbers are given, and no diseases. One imagines that they were all cases of sickness.

This was the hospital to which Hunter was attached, and he was very anxious to become its Deputy Director. There has been considerable doubt as to whether he did or did not get his wish. These recent letters now decide this point once and for all. In the letter, quoted by Peachey, from John to his brother, William, written from Lisbon on July 25, 1762, Hunter says : "When I received your letter at Belleisle informing me that the Secretary of War had promised me the Deputy-Directorship, I was in hopes of getting it ; and when I came to Lisbon Mr. Young told me that I was the person. I had no sooner heard this than Mr. Maddox (one of our surgeons) produces a warrant for the employment granted by my Lord Tyrawley ; and at the same time Lord Loudoun's promise that he should keep it."

Hunter goes on to say that on inquiry it was only granted, conditionally, if he did not come from Belleisle and that Maddox gave up all pretensions to it. He says that he expects that they want to cheat him out of it, as Lord Tyrawley and Mr. Maddox are going home in the same ship. Lord Tyrawley did go home, probably on account of age, and Mr. Maddox was sent home with him to look after him and he returned later.

Now comes an enlightening letter from Mr. Young, the Director, to Lord Loudoun.

LISBON, July 19, 1762.

MY LORD,

I give your Lordship this trouble at the request of Mr. Hunter who is afraid of being turned out of his employment of Deputy Director. I will not presume to make myself a party in this affair, only just to let your Lordship know that he was appointed to that employ in England.

Mr. Maddox yesterday evening showed me Lord Tyrawley's warrant appointing him to that post, but added withal (by his Lordship's order) that if it was already filled up the Warrant was not to take place. I desired him to tell my Lord that it was filled up in favour of Mr. Hunter.

Mr. Maddox told me this morning that he had reported faithfully my answer to Lord Tyrawley and further told Mr. Hunter and me that my Lord was satisfied and that he only meant the warrant should take place in case Mr. Hunter should be ordered to remain in Belleisle.

I am, with profound respect
My Lord, Your Lordship's most
devoted and obedient servant

Rt. Honble E. of
Loudoun, etc., etc., etc.,

W. YOUNG.

On July 22, Mr. Maddox writes the letter reproduced below, which was received by Lord Loudoun the same day and docketed without comment.

SIR,

I shall esteem it as a favour to acquaint Lord Loudoun, that as I understand from Mr. Young, Mr. Hunter was approved of at home as Deputy Director to the British Hospital in Portugal. I have delivered up to him all right that I might have thereunto by virtue of a warrant granted me the 1st. day of June last by the Right Honble Lord Tyrawley.

I am, Sir, Your most humble servant

WILLIAM MADDOX.

June 22nd. 1762

To Colonel Cosnam.

At first sight it seems rather unfair of Hunter, after such a generous withdrawal by Mr. Maddox, to say "they want to cheat me out of it", but then one realizes that Hunter would never have seen this letter.

That Hunter did not give up hope of this appointment for a long time is shown by his letter to William Hunter, dated Nov. 16, 1762 (Peachey) in which he says: "I expected to have heard from you about my directorship, therefore have done nothing in it till I should hear from you; but I am informed by the bye that Lord Loudoun must have a letter from the Secretary at War before he will interfere in it".

The facts are that Hunter never got his deputy-directorship and neither did anyone else. The post was not filled, and both Hunter and Maddox retained their rank as surgeon to the hospital until the end of the campaign.

Through the medium of these letters it is not possible to follow the details of the campaign, nor is the writer a fit person to describe them. By inference, however, it is clear that towards the end of the month of July the troops were moving up towards the front and that the men were subjected to various hardships. There is one incident of men dying on the march which called for a report from the officer in charge and a medical statement from the regimental surgeon, which is worth recording for the details it reveals of march discipline of the period and the position of the surgeon. It would appear that the 3rd Regiment of Foot (The Buffs) were moving up, that they had made part of their journey up the Tagus by boat as far as Port de Muge and thence had to march to Santarem. Major Biddulph sent a detailed account of the march. They started at about 6 a.m., and marched at the rate of about two miles an hour or something under. The surgeon and his mates were divided to the front, centre, and rear of the regiment. Whenever any man fell sick the companies had orders to leave a careful man with him. The rearguard, composed of an officer, sergeant, corporal, and twenty men, had charge of all that should drop behind. Having marched about six miles water was scarce and the men choked with heat and dust. The way lay through defiles of deep sand and the men fell down sick so fast that there were not sufficient well men to attend them.

As a result of this march, which does not appear to have been much more than twelve miles, eleven men died, one presumes of heat stroke. Below is given in full the report on the disaster by the Regimental Surgeon.

THE OPINION OF PETER BERNARD, SURGEON TO HIS MAJYS 3RD
REGT. OF FOOT (BUFFS)

My opinion is, that it was owing to the extreme heat of the weather ; likewise to the imprudence of the men themselves who drank immoderate quantities of water (contrary to all advice given them) whenever they were parched with the excessive heat.

The attention paid to preserve them was as follows, As fast as any man dropt, I removed them into a shady place and gave them every physical assistance, such as bleeding, volatile spirits, &c. They were then left under the care of proper persons till such time as the proper carriages could be procured to bring them into Town.

P. BERNARD.

Aviniaga. July 31st. 1762.

A month later, on Aug. 30, Peter Bernard wrote in to Lord Loudoun as follows : " Having been violently ill of a fever and recovering but slowly it is the opinion of Mr. Hunter that I shall not be fit to do duty for a considerable time and that I stand a much better chance of recovering in England. I hope your Lordship will give me leave to go home and shall return as soon as my health permits."

The letter is endorsed on the outside—" Has leave to go to England for his health."

The contents of the letters of August, 1762, show that the main scene of interest was shifting from Lisbon and being focused on Santarem. The reason for this is supplied by the following brief account of the campaign which occurs in *The Military Life of Field-Marshal George First Marquess Townshend*, by Lieut.-Colonel Townshend (from which book also the map is reproduced) :—

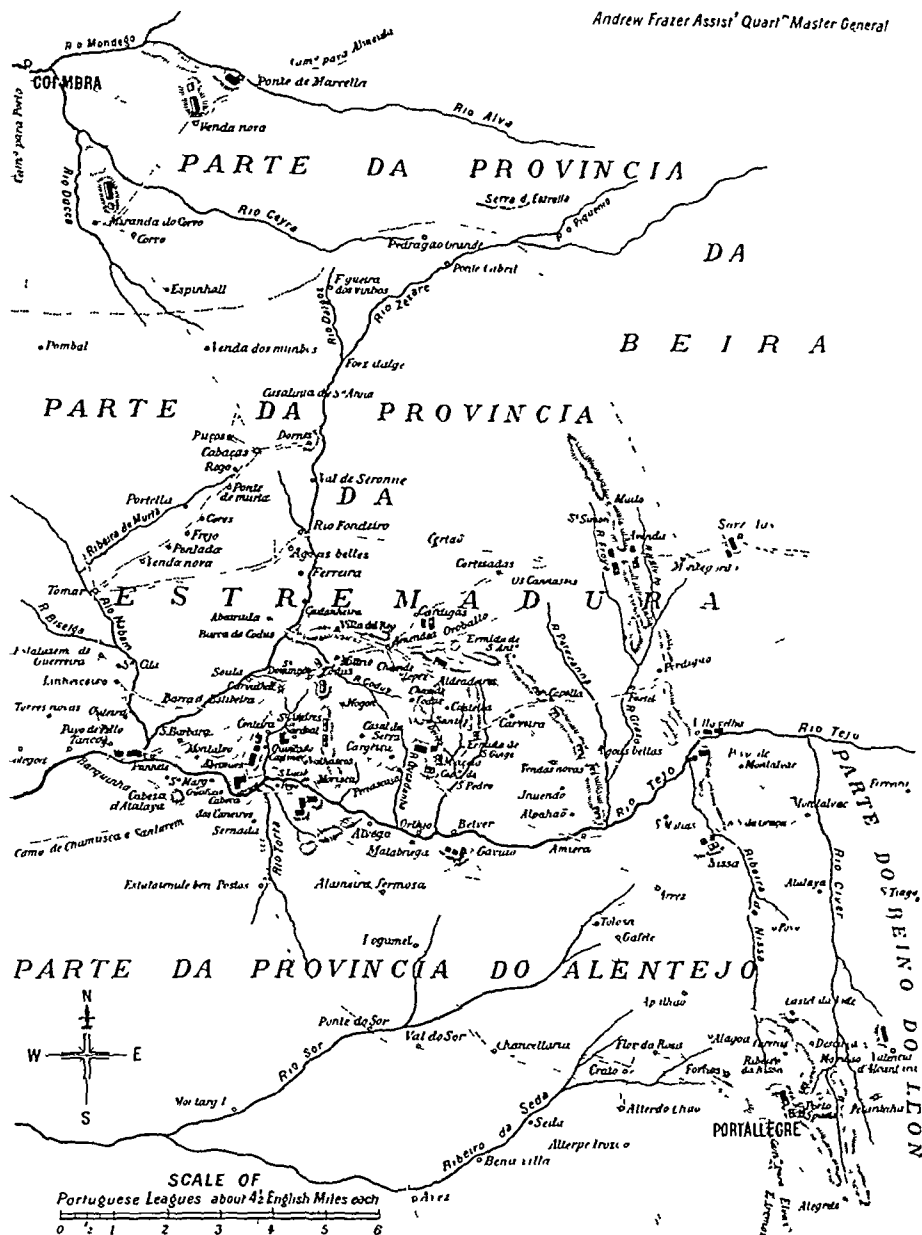
" In August Count de la Lippé had given General Townshend command of a corps in the Beira Alta. His orders were to cover the province as far as possible, preserving the communication with Oporto, for the defence of which he was to construct some works below the River Douro ; he was also to cover the road to Coimbra and especially to watch and prevent the passage of the River Alva at Ponte Marcellas :—

" The Franco-Spanish army was far superior in numbers to the army under Count de la Lippé, who had taken up a strong and judicious position at Abrantes, with the river Tagus on his right and a mountain on his left. As the enemy could make no impression on Marshal de la Lippé's army without driving General Townshend's corps from the mountains called Sierra da Estrella, every movement could be perceived and was reported immediately, and plenty of intelligence was also constantly brought in by large numbers of deserters. Frustrated at this point, the enemy changed their plan of operations to the Alentejo, crossing the river Tagus. On this side the Spanish advanced, the Portuguese falling back . . . the Count de la Lippé clearing the country of all supplies in front of the invaders in much the same manner as Wellington did prior to retiring to the lines of Torres Vedras. Indeed it may be that Wellington had read the account of the operations of the Count de la Lippé's campaign in 1762.

" Want of supplies and transport . . . crippled the Spaniards who having no magazines to maintain them in Portugal had to give up the territory they had overrun, after a few small skirmishes, and retreat across their own frontier on the approach of the winter of 1762. Thus ended the campaign happily for the Portuguese though the prospects of that country had been gloomy enough when war was declared.

A PLAN of that Part of the Country which was the Scene of Operations in PORTUGAL in the Campaign 1762

Andrew Frater Assist^t Quar^t Master General



(Reproduced by kind permission from "The Military Life of Field-Marshal George First Marquess Townshend", by Lt.-Col. Townshend.)

"A general peace was declared in Europe on Nov. 15th, 1762, and all the special service officers and the 5000 British troops which had been lent to Portugal returned to England."

For us the town of Santarem now becomes important as an advanced base and the site of a general hospital, and it was to remain so until the end of the campaign. This town is situated on the right bank of the Tagus, about 46 miles from Lisbon and about 40 miles below Abrantes. A study of the map will show that the British forces were encamped on a comparatively small area in the district between the Tagus and the hilly country to the north of this river, extending into the range of mountains called the Sierra d'Estrella.

As we shall see later, a number of small hospitals were set up, at Tancos, St. Domingos, Mont Alvo, Vendas Novos, to which various members of the staff of the hospital were sent up from time to time. Evacuation to the hospital at Santarem seems to have been by boat, the sick being brought down from the Regimental hospitals to Abrantes or Punhete on the Tagus.

In addition to the British Forces about Abrantes there was a detachment farther to the south-east at Portallegre in the Province of Alentejo. Hunter was stationed here for a time, and several communications from him are concerned with the state of the sick there.

By the middle of August many sick were being sent to Santarem, and as there was no hospital there at that time, Mr. Hugh Smith, the Master Apothecary, was sent to see what arrangements could be made and to report to Mr. Young on the state of affairs. His letter, dated Aug. 13, gives a vivid account of the conditions :—

On account of waiting for boats and carriages it was Wensday morning before I arrived here and finding the sick in want of necessities of all sorts and very near half of them laying on the ground I have since hurried in providing bedding and proper accommodation for them which has prevented me from sending you a return before this. The hospital, which they are in, is an indifferent one and only contains about 120. Pursuant to his Excellency's Lord Loudoun's orders I have looked for another hospital and I have found a most elegant one for that purpose which will accommodate 300 sick, it was a Jesuits Colledge. . . . The disorders of the sick are in general fevers and fluxes, some of the putrid kind. . . . I should be glad you would send down some of the bedsteads which are at Lisbon as we have no such thing here. . . . Upon enquiry I find the sick can be carried by water to this town from the Army in six or eight hours and as the situation is on the top of an hill and well supplied with water I think it is the proper place for to establish a general hospital.

By the same post Mr. Smith sends a return of the sick, showing that there were 93 men to be cared for. There is no mention of wounded, but he refers to a considerable number of malingersers.

By Aug. 28, Director William Young evidently found it necessary to come up himself, and on that date he sent a report of the hospital at Santarem to Lord Loudoun. From this report it is clear that considerable additions had been made since the Apothecary wrote on the 13th. The letter is also illuminating as to the difficulties he had in dealing with the local authorities.

STATE OF THE HOSPITAL AT SANTAREM

Old Hospital	101
New Hospital	59
Great Hospital	240
	—
Totall	400

These are much crowded there being proper and convenient room for no more than 250.

They come in (except a very few) without tickets or a list of their names or numbers. . . . The corridore is very remiss in sending in firing and straw. When I came I found many lying on the bare ground. When he does provide anything it is with so much sending after an solicitation that . . . it becomes very inconvenient to the hospital and the service suffers. . . . As to the men they lye pretty well. The wards are as clean and their cookery as well conducted as it can be at this juncture, and upon the whole they are pretty comfortable. The great and only material defect is want of room. As to the rest they have been (today) pretty well off, for a quantity of straw came in.

The mates from Tancos send a very bad account of the hospital there. Mr. Hunter is gone up. There were 130 and men coming in daily.

When the men are in a convalescent state are we to put them in Quarters, if so has the Corrigidore orders to give Billets. Are they not then to be struck off the Hospital Books?

Seeing that there were expectations of action, the hospital at Lisbon was depleted of its staff. There is a footnote (*Letter 132*) which says, "Mr. Hunter being sent up to the army as there was an appearance of action, there remained only one surgeon to the two hospitals for which reason Wm. Young, Esq. was appointed a surgeon of the hospital the 6th Sept. 1762".

It will be remembered that there were only three surgeons on the staff of the hospital, namely, John Hunter, Francis Tomkins, and William Maddox. Maddox had gone back to England looking after Lord Tyrawley. Hunter had gone up to the army, so there was only Tomkins left and he went up later also.

Who was this William Young who was appointed surgeon on Sept. 6? Were there two men of the same name, or was the Director, William Young, given two offices? Johnston suggests in his Roll that the Director was succeeded in his office by another man of the same name. We shall see that in all probability this was not so, but that the Director was given the extra post. The evidence for this is as follows:—

Lord Loudoun must have sent an order to the Director, telling him to act as Surgeon when necessary, for on Sept. 8 William Young writes: "I shall cheerfully obey your Lordship's order to act as Surgeon when and as often as my other Business will let me".

Then on Oct. 23, 1762, comes this letter:—

MY LORD,

Nothing can equal the satisfaction your Lordship has been pleased to give me by my new Commission but the gratitude I feel towards my noble Patron. I most humbly and heartily thank your Lordship.

This most pleasing and expressive Testimony of your Lordship's approbation of my conduct came unsolicited and was never in my thoughts; now it will never be out of them.

I am My Lord
Your most grateful
and most obedient
humble servant

Rt. Hon^{ble} E. of Loudoun,
etc., etc., etc.,

W. YOUNG.

There seems no doubt, therefore, that there were not two William Youngs, but one William Young, and that Lord Loudoun combined in him the posts of

Director of the Hospital and Surgeon, even as he had done in North America when James Napier was, at one and the same time, Director of the Hospital and its chief Surgeon.

The month of September, 1762, seems to have been a trying one for the medical service. Sickness among the troops was rife, accommodation for the sick was poor, and there were expectations of active fighting, which, however, do not seem to have materialized. Reports of many sick kept coming in from camps and temporary hospitals. Many were the complaints that men came down the line without proper details and descriptions and the lists furnished were almost always without diagnoses. One perfect return, however, was made, signed Donaldson, Mate to the 83rd Regiment. In this return it is stated that he had 54 sick, of whom 43 were in camps and 11 at Abrantes. All the diagnoses were most carefully set out, and from it we can gather what were the prevailing disorders. Ague, flux, and fever, the old well-known enemies of soldiers, were the most common complaints. Not a word of a wound.

Added to this return is an interesting item, namely, the recommendation of four married women as nurses, namely, Mrs. Hawkins, Mrs. McGaw, Mrs. Clack, and Mrs. Matthews. With the exception of Hawkins, none of these names appear in the official list of the nursing staff of the hospital. It seems likely that these ladies were volunteers from the married women who followed the fortunes of their husbands in the army.

At this time there seems to have been a prospect of fighting, and Hunter was kept busy. He had been sent up to Tancos, a small village not far from Abrantes and just behind the line.

The following letter from the Director to Lord Loudoun, dated Santarem, Sept. 8, 1762, explains the state of affairs :—

I just received the honour of your Lordship's commands of the 5th. inst. Mr. Hunter communicated to me the letter you sent him at Tancos, requiring him to break up the Hospital there and to joyn the army with what mates he could get together after making a proper disposition for the removing the sick both at Tancos and this place. Your Lordship in your letter was pleased to require that I should send all the assistance I could to Mr. Hunter. Most of the mates were at that time with the Army as appears by the enclos'd return of the distribution of Mates, and as we had but four of them with us, and upwards of 600 patients so it was not possible to spare one, but I sent medicines and immediately despatched a express to Lisbon for more hands . . .

I have a letter this day from Mr. Hunter at Tomar (dated the 5th. inst). He does not downright say he is sick but that he is allmost knocked up. He has had much to do. He says he is stopped at Tomar for want of a carriage, but hopes to set out soon.

At the same time Mr. Young sends a return of the sick in hospital at Santarem. There were 694. In a previous letter he had said that there was accommodation there for 400, so there is no wonder that there were complaints of overcrowding. In a footnote to his sick return Mr. Young adds :—

N.B. The distempers in general are not very bad. In the last twelve days we have lost 25, several of which were brought just dead out of the boats.

Five days later Mr. Young reports that they are getting straight in the hospital at Santarem, and that as he had been able to discharge 130 men the overcrowding was not so bad. He presses the Commander-in-Chief to let him know in good time should he want a hospital moved in a great hurry up to the front line. He

says he has much difficulty in getting transport: "I wish we had a small number to ourselves with a guard over them to secure them from being pressed, indeed I think if a battle should happen there would be a good deal of distress . . ." Here is the age-long cry of the medical service to have its own transport. The Casualty Clearing Stations made just the same request during the late war. Transport always was a difficulty.

He also reports the dangerous state of one of the surgeons' mates, Mr. De Veil. He died on Sept. 26.

By Sept. 15 Hunter was at Coimbra. He seems to have accompanied Lord Loudoun, for the purpose of attending to the sick from General Townshend's Division, whose duty, it may be remembered, was to keep open communication with Oporto and to protect the crossing of the river Alva at Ponte de Marcella. This is his letter, so lucidly expressed that one may follow at once the needs of the situation:—

MY LORD,

When I returned from Venda Nova I immediately waited upon Mr. Nash, to get his assistance in procuring what was necessary for the removal of the sick. When I told him your Lordship's pleasure he found the following difficultys, viz., that it was very uncertain, the getting a vessel fit for the purpose at Figueira. When got, that it would coast above fifty Moidores: that it might wait some time for a Barr: and on entering the river Tagus might be embarrassed by the Health Office. If these are not sufficient objections I shall still pursue the first plan. Mr. Nash is at Figueira at present; therefore it will be necessary to have an order from your Lordship to press a ship, if she is to be found there. If your Lordship did not think it tedious, a ship might be ordered from the river Tagus, to Figueira, viz., the Hospital Ship from Belleisle. Or if your Lordship was not desirous of having them at Lisbon immediately I dare say that they might all be removed by land in a fortnight's time; for above one half will be able to walk to Santarem, if their accoutrements are carried and slow marches; and the others will be able to do it with a little assistance from carriages. They are all so well that one Mate is sufficient to take care of them. If your Lordship should approve of this I should be glad to know what is to be done with those men of General Lambert's Rgt. that may be fit to join their Regts by that time. The sick from Ponte de Marcella are not yet arrived.

Coimbra

Aug. 15th.

N.B. The Ship's name

is the *Betty* Transport
Captn. Pescote Master.

My Lord I am with
Great Respect your
Lordship's Most obed^t
and Humble Servant

JOHN HUNTER.

(N.B.—This letter is dated by Hunter Aug. 15, but there seems no doubt from the endorsement on the back by Lord Loudoun that this was an error, and that he meant Sept. 15.)

To this letter Lord Loudoun replies from Miranda de Corvo on the same day, Sept. 15, 1762:—

SIR,

You are hereby directed upon the receipt of this Order to hire Vessels proper and sufficient to transport the Hospital and Baggage now under your care and direction at Coimbra to Lisbon, for which purpose I shall make immediate application for Convoy.

To Dr. Hunter.

If the hospital were having a difficult time it is no less certain that the surgeons with the army were equally hard pressed, and a sample of what they had to contend with is shown in a letter addressed to General Armstrong and dated Cabassa, Sept. 20, 1762, from William Wiseman, Surgeon to the 75th Regiment.

SIR,

I'm left here with twenty nine of your sick, eleven of the 75th. and one of Lord Blaney's, Being in a melancholy situation to get provision for them, have took the liberty to detain Nicholas McColly of the Major's company in your regiment to interpret for me, that I may get them to Santarem as soon as possible, otherwise the poor sick must inevitably suffer through want of common sustentances.

This morning I sent a corporal and two men with a [indecipherable] to get sheep for the sick, instead of getting them he was threatened by the peasants and a Portuguese Dragoon would have killed him if he had not had a stick to defend himself. This I mention that you may represent to his Excellency, the Earl of Loudoun, what a miserable situation we are in.

There being now need for more hospital accommodation, and that it should be nearer the line, Lord Loudoun sent orders for a hospital to be opened at Abrantes, and Mr. Young immediately busied himself sending up stores for 200 patients and calling on the hospital at Lisbon for four supernumerary mates. The work at Lisbon was obviously light at this time, for the number of sick was reported as only 50, while at Santarem there were 508 (October, 1762).

During this time it would appear that there were considerable movements of the troops, and that the members of the medical service found it difficult to keep them adequately supplied, and an interesting letter comes to hand, signed both by John Hunter and Francis Tomkins (of whom more anon), dated Macoa, Oct. 7, 1762. In this a representation is made regarding a temporary hospital, and later on we shall see that Hunter sends in his ideas of what such a hospital should consist of.

MY LORD,

As there is no provision made for the Flying Hospital, as in other countrys, we think it incumbent upon us for the better and more speedy carrying on His Majesty's service to acquaint your Lordship of the necessity of having mules or horses, to transport our mates, medecines and instruments, many of them have fallen sick from the fatigue and want of these conveniences and also many of the medecines and instruments have been obliged to be left behind. Therefore we hope your Lordship will see the propriety of this application and allow us for each surgeon two beasts and three more to be in common for mates that attend the Flying Hospital.

We are and with great respect etc., etc., etc.

Owing no doubt to Hunter's representations of the difficulty of looking after the large number of sick, he was asked to give in writing a plan for a hospital such as he thought was needed. He does it briefly and lucidly, and on reading it one can realize at once the needs he saw and his idea of overcoming them.

PROPOSAL FOR ESTABLISHING AN TEMPORARY AT PUNHETE*

- 1st. It need not be a General Hospital but only a half way one between the Flying Hospital with the Army and the General Hospital at Santarem.
- 2ly. That it should contain above one hundred men.
- 3ly. That three carts would be sufficient to transport all the sick as they fall ill, if rightly managed, from the Army to the said hospital, if the Army keep near this place.

* Punhete is on the Tagus, a little downstream from Abrantes.

- 4ly. That a boat might be kept at Santarem for the transporting of such sick to Santarem as were not likely to get soon well.
- 5ly. That the convalescents at Santarem might act as guards to the boats and hospital. The advantages arising from such an Hospital :—
 - 1st. The sick would be shorter in their passage than as from Abrantes.
 - 2ly. Slight cases would get well without further carriage therefore sooner of service to the Army.
 - 3ly. Bad cases would be better taken care of and not run that risk of their lives that they have hitherto done.
 - 4ly. It would be a proper exercise for the convalescents.

It appears this excellent suggestion was not acted on.

Sickness was not confined to the troops at the front, for Dr. William Cadogan, the senior physician to the Hospital, who was left at Lisbon looking after the sick there, now falls ill ; and on Oct. 21 he writes this piteous letter to Lord Loudoun, asking leave to go home :—

MY LORD,

I am at last forced to do what I have long struggled to avoid troubling your Lordship with my miserable condition. I have been extremely ill this long while, about ten weeks ago I was seized with a violent flux which lasted a good while after that a severe fit of the gout in both my feet a disease new to me, this confined me to my bed and broke me all to raggs. . . . However I was beginning to recover when I was seized with the most malignant fever that ever was. . . . I cannot recover having yet not the least appetite nor natural rest. I am daily and hourly sinking with more pain and weakness so all that remains for me now is to throw myself upon your Lordship's compassion and humanity hoping you will be so good as to give me leave to return to England as the only means left me all other help having been tried in vain to preserve the miserable remains of life I have left.

Dr. Cadogan got his leave home and Lord Loudoun wished him a speedy recovery, and it is gratifying to record that he got quite well, living to the age of eighty-six, that he attained honourable office in the Royal College of Physicians, and that his portrait hangs now in that College (Munk's Roll).

There was now a vacancy as physician on the Hospital staff, and it is amusing to see the applications which were made to Lord Loudoun for the post. A Dr. J. Cantley wrote to him twice asking that he might be appointed. I do not know who Dr. Cantley was, but suspect that he was a private practitioner resident in Lisbon. There is no reference to him in Johnston's Roll, or Munk's Roll, nor was he, as far as the documents at our command go, attached to the army. Then John Hunter also applied.

If one turns to Peachey's *A Memoir of William and John Hunter*, in a letter dated Portallegra, Nov. 16, John says to William : "I am now applying for Phisician, if I can get it I shall be a Dr. as well as the best of you. I have attended his Lordship in all his marches, and have reason to believe that I am well with him." Here is a copy of the letter Hunter sent to Lord Loudoun on Oct. 28, 1762* :—

MY LORD,

I take the liberty to throw myself upon your favour. I have been informed that your Lordship has given Dr. Cadogan leave to return to England. I therefore presume that a Phisician will be wanted in that hospital. If so I take the liberty to recommend

* This letter is in the possession of Dr. Fenwick Beckman, of New York, and is reproduced in *Annals of Medical History*, New Series, Vol. 8, No. 4.

myself and I hope that the following reasons will plead some excuse for troubling your Lordship. First, my Lord, I have the oldest commission on this Staff. In the second place I came here with the promise of my ten shillings a day being continued, which your Lordship know I have not got. Thirdly I have your Lordship's promise which you was pleased to give to General Lambert (upon the Cop of the above) that your Lordship would serve me the very first opportunity and desired that he would let you know it. I hope that my education in Phisick will be no objection to me.

I know that your Lordship has natural objections to the increasing the hospital expences but I hope that those can be done without any additional expence for I should not have the least objection to my acting as surgeon in the same hospital or indeed in any capacity that the Service required, as I have done all along since I came to Portugal.

Very rightly Lord Loudoun did not wish to lose the services of a good surgeon with the army, and so Hunter was not given the post, nor was Dr. Cantley.

Another applicant for the post was Mr. Walter Hamilton, the second Apothecary to the Hospital. He wrote on Nov. 8: "I beg your Lordship will consider me, who have been eighteen years in the Service constantly attending my duty, which I am certain very few can say. . . . I have been so reduced by a long fever which I got attending in the Hospital that I am not able to write but hopes your Lordship will excuse that at present." He did not get the post either.

On Nov. 9 Dr. Cadogan wrote thanking Lord Loudoun for giving him leave to go home, and tendering his advice as to what should be done. He said that during his illness the work had been done so well by Mr. Smith, Mr. Hamilton and Mr. Golding, the best of the mates, that he did not think there was any necessity to fill his place, and he ends his letter: "I must therefore beseech your Excellency not to let the crows eat me before I am dead".

The vacancy was filled eventually by the appointment of Richard Huck. Huck was an old friend of Lord Loudoun, for he was a surgeon of the general hospital in the expedition to North America, and sailed with him in the same ship. When Loudoun had his headquarters in New York, according to Pargellis, "Every evening, except when his hospitality was repaid elsewhere, there dined with him at the long table, heavily weighted with silver, his intimate 'family' made up of his Aides-de-Camp, etc. . . . Doctor Richard Huck, the gay and learned, who had been surgeon in Loudoun's Scottish Regiment in the '45 was often there."

Later Dr. Huck became a Fellow of the Royal College of Physicians and Physician to the Middlesex and also to St. Thomas's Hospital. In 1777 he married an heiress, changed his name to Saunders, and became the father of the future Viscountess Melville and the Countess of Westmoreland (*D.N.B.*, "Johnston's Roll", "Munk's Roll").

Now that autumn had come the Commander-in-Chief had to begin his preparations for the coming year's work and to send back to England for all things necessary, and as he had to make arrangements for the medical service as well as the purely military, he sent in a request for lists of medical stores which might be required. Director William Young was ordered "to send me immediately an account of what drugs, necessities, assistance and every other thing you think may be requisite in your Department".

These lists, signed by William Young, are included among the letters, and are an interesting commentary of the furnishing of the hospitals at that time. Among the articles asked for were:—

Five hundred complete sets of bedding (each set containing one palliasse, one bolster and one pair of sheets, one blanket and one coverlet). Twenty pewter bed pans, twenty-five chamber pots, thirty bleed porringers and twenty-four tin candlesticks and extinguishers. Of turnery ware asked for, were 200 white platters, 150 quart bowls, and five gross of wooden spoons. Then came a request for stationery, with the addition of a few quires of gilt paper. The reason for this last demand does not appear. After these stores follow a long list of drugs and a much smaller list of instruments. These latter include :—

Two sets of amputating instruments complete ;

Two sets of instruments for the Trepan ;

Two dozen of dissecting knives ;

Twenty dozen lancets ;

Six dozen bag trusses and four dozen spring trusses.

Attached to the list of instruments is a little note : “*N.B.* The last scissors and lancets were very bad, it is desired Mr. Bodker be no more employed.”

By the middle of November it would appear from the various letters that active military operations were for the time being at an end, and that all the army had to contend with was sickness, which was considerable. It is evident also that Director William Young was greatly concerned with the amount of very sick men with whom he had to deal, and that he was not satisfied with the way they were being handled. The letter he wrote to Colonel Cosnam, the Adjutant-General, on Nov. 6, indicates that he was fulfilling the function of a Director-General of Medical Services and giving the Executive Branch some sound advice. He says :—

I hope that if the Army should continue quiet we shall have fewer patients sent to the hospital ; indeed if I may presume to offer my advice I think it were better to send us none at all but to let every Regiment take care of their own sick for some time. Among other reasons for this measure, the injury the men suffer in being brought to the Hospital is a material one. Our Gentlemen might be dispersed to the different cantonments and find employment in assisting in the regimental infirmary. This I only mean to take place while the Army has a prospect of remaining quiet. If you think what I propose is reasonable you will please communicate it to his Excellency and at the same time ask him if he thinks it necessary any longer to keep up the Hospital in Lisbon.

The transport of the sick and the hardships and indeed injury to which they were subjected was a constant source of anxiety to the Director, and several of his letters dealt with this point. Though there is no letter from Lord Loudoun concerning Mr. Young's suggestion it seems that it was adopted, in part at any rate, for one finds indications that there were a number of camps in which comparatively small numbers of sick were held, and from which sick returns were made, and to which members of the hospital staff were attached or at any rate visited from time to time. John Hunter certainly visited quite a number of these camps and sent from them returns of the sick. Never once, however, was there a mention from Hunter of having to treat a wound.

We must now interrupt the story of the campaign for a moment to consider the doings and characters of some of the actors.

What follows may seem trifling tittle-tattle and scarcely worthy of print and paper, but it really concerns ours hero, John Hunter, and that is the excuse.

Students of Hunter will recall that Jesse Foot wrote *The Life of Hunter* in 1794. Jesse Foot had a mischievous pen and wrote of Hunter in the most malignant manner. He made out that he was an unpleasant individual, and alleged that while in Portugal he quarrelled with a colleague, Francis Tomkins, and that their difference was so great that a sword was actually drawn. The following is the extract from Jesse Foot (p. 80):—

He had scarcely arrived at Portugal before he excited an uneasiness among the faculty, which their situations had never experienced before. He turned the common intercourses of social good humour into suspicious tauntings of jealousy; he created a faction and a consequent disgust. This brought on an explanation from one who was his senior in the army by ten years;—from one who had been a faithful follower of the fortune of the Duke of Cumberland, and had dressed his wounds in battle: he was roused to draw his sword on John Hunter, which was sheathed without the quarrel being reconciled—for what reconciliation can remove suspicion?

The confirmation of this I am not disposed to doubt, but some there are who may: those I will assist as far as it is in my power by assuring them,—that the manly veteran, Tomkins, of Park Place, is very capable of explaining the fact.

Francis Tomkins was one of the surgeons on the staff of the hospital. There are a number of letters from him in this new collection, which tend to shed a light on the accusation by Jesse Foot and incidentally reveal that Francis Tomkins, himself, was of a fiery disposition.

The first incident concerns a certain Samuel Hayes, and a quarrel between him and Tomkins in which Hunter was also involved. Samuel Hayes was one of the surgeon's mates attached to the hospital, and in the month of November, 1762, was stationed at Mont Alva, where there was a subsidiary hospital. He had been put under arrest by Tomkins, and evidently Hunter had been sent by Lord Loudoun to set him free. Hayes writes a long and detailed account of the affair to Lord Loudoun in which he justifies himself and accuses Tomkins. The letter is too long to put out in full, but the relevant passages are quoted below:—

MY LORD,

In consequence of the honour of your Lordship's commands by Mr. Hunter I came out from the arrest I had been put under by Mr. Tomkins and entered again on duty.

He then says that as an arrest is an imputation of guilt and as he can only look to Lord Loudoun for justice he wishes to explain the circumstances. Then he tells the story:—

On Thursday the 4th. of this month (Nov. 1762) while I was unpacking the medicine chest and two other large parcels of medicines . . . Mr. Tomkins sent to me by a soldier from the next room for about four doses of Dovers powders. The soldier brought a single piece of paper in his hand for it in which I gave him the powders. I, not knowing what Mr. Tomkins intended to do with them, did not fold it up. Mr. Tomkins apparently was very angry at this, said he wanted the powders made up in separate packets, and made some injurious remarks.

Then Hayes goes on:—

On Friday afternoon sitting with Mr. Hunter and Mr. Tomkins I was complaining of having suffered a good deal from not having lain in a bed or had my clothes off for near six weeks (having lost my baggage in my hurry to return to the army from Almeida) I told Mr. Tomkins it was unkind of him to have deprived me so long of the bed Mr. Young had sent me and now that it was arrived to take from me the blankets I had given a receipt for.

In consequence of this he flew into a great passion—used language unfit for an English Doctor to have born with, called me rascal and rose from his chair in great fury, caught a quart bottle from the table, came very near me, shook it at me and threatened to knock me down. As I had been accustomed to his passion I knew how far his discretion would carry him and sat still and behaved with great coolness. He immediately after this sent me to my room. Mr. Hunter was present during this and tho' he would avoid interfering in matters which that did not absolutely concern him yet as a man of integrity and honour will not refuse when asked doing me common justice.

He goes on to say:—

Lest your Lordship should imagine from this extraordinary behaviour something on my side culpable I must beg pardon for mentioning to your Lordship that he has quarrelled in almost the same manner with the whole Hospital, viz, Dr. Cadogan, the two apothecaries, Mr. Hamilton and Mr. Smith, and since he joined the army with Mr. Hunter very seriously several times . . . ”

What are we to make of this after all these years? There is no comment by Lord Loudoun, or by Hunter, on this letter, but the fact that Hunter was sent to free Samuel Hayes from arrest makes one think that he had probably talked to the army commander and told him that there was nothing in it. Hayes says that it was Tomkins who was quarrelsome and not Hunter, and there is another incident between Tomkins and another mate which seems to bear this out.

J. W. Kingston was one of the mates and he had been sent to look after the Count de la Lippé and his family. Francis Tomkins evidently had his knife into him, for he reported his conduct and impertinent language to the Adjutant-General, Colonel Cosnam, in a letter dated Dec. 16, 1762. He said that Mr. Kingston refused to look after the sick of the army because he considered that he was in the service of Count Lippé, and when he was ordered to do so used impertinent language. He also implied that Mr. Kingston feigned illness whenever there was work to be done, and that his ill conduct was notorious. By the same post he encloses a letter which Mr. Kingston had sent him which gives his side of the story. This is as follows:—

To Mr. Tomkins, Surgeon.

SIR,

I must say you were illnatured enough to use expressions today which I should be far from using to the most abject soldier, that was to say I always fell ill when there was anything to do and that too before a number of officers.

Were it not notorious that I did everything in my power during the whole campaign for the good of the service and omitted no opportunity of serving those in distress it would give me some uneasiness, but as I am conscious I am not, nothing more remains to be done but to clear myself from any imputation your repeated insinuations are likely to cast on me undeservedly.

I must therefore insist upon your letting me know the authors or author for such expressions as I am not to be treated at this time of day in a manner so unbecoming a gentleman. If you do not you may depend I shall take all proper steps to justify myself, so am with what respect is due

Yr. Hum^e Ser^t

J. W. KINGSTON.

Both the letters, that from Mr. Tomkins and also from Mr. Kingston, were sent up to Lord Loudoun, and on Dec. 19 we find a letter from the Adjutant-General saying that his Lordship was surprised at Mr. Kingston's behaviour, that

he did not approve of it, and in other words that he was not to do it again or he would have to take notice of it.

There is still one more letter about this affair from Mr. Tomkins to Colonel Cosnam :—

I received yours relating to Mr. Kingston last night and shall communicate to him the contents. He sent me a message since my last that he wanted much to go to Lisbon as he's had a clap up on him these two months past. In short this is but a trifling excuse for the rest of the mates say it is of no consequence being scarcely to be called a gleet. Therefore I will recall the mate I left there and order him to his duty, tho. it cannot be called a duty as he has so little to do.

That there had been some sort of a quarrel between Tomkins and Hunter is clear from the following letter, dated from Abrantes, Dec. 6, 1762, from Tomkins to the Adjutant-General, with whom he seemed to be on friendly terms :—

As to Hunters differences and mine it only proceeded from a jealousy of my having made interest with his Lordship to stay behind ; which upon my honour I never did and that you well know. When I met him that morning he expressed himself in that light, now it was always well known that I was not that sort of person, and I should be glad he was convinced to the contrary.

That is all that is known about the matter, and probably all we ever shall know. From the evidence before us it would seem that Mr. Tomkins was a quick-tempered and not very tactful person, to say the least of it. One does not know how Jesse Foot came to hear of it, and there is certainly nothing to show that Hunter was in any way to blame, though it may have been that he was not an easy man to get on with.

Of course this all seems very silly now. One must remember, though, that in times of stress, when the conditions are bad, when "fed up" and the exaltation of battle has died down, these little troubles become magnified. Many of us can remember during the late war, how at times nerves became frayed and tempers short. So we must not be too censorious when we read of these bickerings of long ago, and pass over them with an indulgent smile.

December, 1762.—Returning now to the medical affairs of the campaign, it should be remembered that a general peace was declared in the middle of November, 1762, although the Treaty of Paris was not completed until the February of the next year. So there was no more fighting, but there was still a great deal of sickness. As was stated previously, the scene of the medical activities had shifted from the base hospital at Lisbon, though this was still kept open, to the hospital at Santarem, where what would amount to a base hospital was established. Writing from here on Dec. 2, 1762, the Director, Mr. Young, says : "We have been very sickly here for some time past and a very bad fever now prevails which carries off a good many men". He also emphasizes his old complaint that the men suffered too much in their transit from the camps to the hospital : "In a former letter I took the liberty of remarking that the sick suffered much in being transported from one place to another. We have sadly experienced the truth of this in the three last parties of sick that came down to us making in all 133, six of whom died before they reached the hospital gate."

Transport of the sick in time of war is always a difficult problem. Ambulances had not yet been invented, and the removal of the sick would be by means of

country carts filled with straw and hired for the purpose. In this campaign it would appear that the method adopted was to bring the men in carts to the nearest convenient point on the Tagus and thence by boats, probably open boats, to the hospital at Santarem.

Together with the above letter, the Director sends in the hospital state from Santarem. From this it can be seen that at this time they had about 500 sick in hospital. It seems difficult at first to know what was done when the men became well enough to leave hospital but were not fit to join their regiments. There were no convalescent camps, but it seems that, instead, the men were quartered out in billets in the town and put under the charge of a combatant officer, who arranged them in messes and was responsible for their welfare and discipline.

There are a number of returns of this nature from Lieutenant McManus, of the Buffs. These returns show that there were often from four to five hundred men who were in quarters.

Subsidiary Hospitals.—Then it is quite clear that there were a number of smaller hospitals nearer the line, and that various members of the hospital staff looked after them.

There was such a hospital at Mont Alvo, and we know that Francis Tomkins was there, for on Dec. 5, 1762, he wrote the following letter to the Adjutant-General, Colonel Cosnam :—

SIR,

Enclosed is the return of the sick (113) and as some men recover fast I hope in a little time to remove the whole to Santarem. Since my last return I've sent to Santarem 28 men and have been applying these two days at Punhete (which is the most convenient place) for another boat, and am promised that I shall have one to-morrow when I shall send between twenty and thirty more to Santarem. We have now here about 25 recovered men that I should have sent yesterday to Abrantes where the convalescents from Santarem are very sickly. Numbers have been sent back but Capt. Sutton tells me he intended to acquaint his Lordship of the sickly state of the town, people who die very fast; it is certainly a most unhealthy place for convalescents and to try if his Lordship would order them to be removed to Tancos or some other good airy place.

I must beg that you all let me know how I am to proceed with regard to myself and stores. I would willingly, between friends, when I evacuate this hospital to get to Lisbon if it was possible at least I should be sorry to be ordered to join the army at this time, that I might not run the risk to be left behind with the sick at Portallegre or some cursed place among these Portuguese. Will you give me a line as soon as possible that I may know where I'm to go. I believe I shall be able to get the whole from hence in ten days at least.

I am Dr Sir

Yours sincerely,

FR. TOMKINS.

At the same time John Hunter was at Portallegre, the place Tomkins did not want to go to. On Dec. 7 Hunter sends a return of forty men who are to go from Portallegre to the general hospital.

The complaints made by the Director of the Hospital that the lives of men were endangered by being transported according to the orders of the Commander-in-Chief were very sympathetically received. There is a letter to him from the Adjutant-General to that effect in which he says that they were never to think of transporting men whose lives were in danger. By the same letter came the following strange request, which was at first difficult to understand :—

As you do not mention I fear you have either overlooked or not received my letter desiring you to get a quantity of acorns collected from the cork trees for my Lord. It is not yet too late and he expects about two bushells entirely of the large cork tree of which there are numbers about Santarem.

To this Mr. Young replies on Dec. 16 :—

I did not forget My Lord's Com^{ds} about the acorns. I am told the finest sort are about Elvas. I am getting the best that can be got hereabouts.

Then Mr. Young writes again on Feb. 17, 1763, to Colonel Cosnam :—

Doct^r. Morris desires you will present his Respect to My Lord and acquaint him that he put four basketts of slips of the cork tree in a boat procured for that purpose last Saturday morning and that he gave them in charge to Mr. Hayes Surgeon's Mate.

This reference to acorns and slips of trees was puzzling for a time. What did the Commander-in-Chief want with acorns? Reference to the *Dictionary of National Biography* supplies the clue. Lord Loudoun was fond of forestry and a lover of trees, of which he planted many on his estate at Loudoun Castle in Ayrshire. Reference, moreover, to the life of a contemporary of Lord Loudoun—namely, William Augustus, Duke of Cumberland—by Charteris, records that planting then was extremely in fashion. A great deal of the planting in Windsor Forest was done by Cumberland. "A knowledge of trees and planting had become as necessary a part of a polite equipment as the lighter accomplishments of sport and gambling." So Lord Loudoun, in gathering acorns in Portugal, was only following the fashion of the day. It would be interesting to know if cut trees gathered by the medical staff in 1763 now grow and flourish in Ayrshire.

The hospital at Mont Alvo was being closed down, and Mr. Tomkins, as we have seen above, was anxious to know what he was to do, and he also seems to have been concerned with the danger of transporting the very sick men. On Dec. 12 he wrote to Colonel Cosnam :—

I received yours at Montalvo last night and I have come here (Abrantes) this morning to prevent Mr. Kingston from sending any more bad cases. I assure you none went from me, but was in a convalescent state to Santarem and those were provided with bread and wine as also a nurse altho there was a probability of their getting down in a few hours.

A nurse, let it be remarked, went with these patients. This is the only reference to a nurse being employed on duty away from the hospital.

In addition to all his other duties, Lord Loudoun, as Commander-in-Chief, had to receive and deal with all requests for promotion. The following letter illustrates this point, and it is of additional interest seeing that it comes from Mr. Home, whose daughter John Hunter later married. It runs as follows :—

Mr. Home Surgeon to the 16th. Regiment of Dragoons begs the favour of Brigadier Fraser to make his most humble comp^{ts} to the Earl of Loudoun. He received a letter about the time of the Regiment embarking for Portugal informing him that Mr. James Pringle had been so good to recommend him to his Lordship's favour and Protection in case a Vacancy should happen in the Hospital; but it has been his great misfortune that it has never been in his power to pay his respects to his Lordship.

Mr. Home was informed in England that he would have an Allowance of Baggage for carrying his Medicine Chest, but he has not yet received any, tho' the Surgeons to the Regiments of Foot have long ago, if his Lordship has any doubts concerning his right to it, Coll^e Chancey (he is told) is the properest person to inform his Lordship what is the establishment in Germany.

This is endorsed by Lord Loudoun without comment.

On Dec. 24, 1762, Mr. Maddox, one of the surgeons of the Hospital, reports his arrival.

MY LORD,

I have taken the Liberty to inform your Lordship of my Arrival here Yesterday, in His Majesty's Ship *Assistance* after a Passage of Thirty three days and to acquaint your Lordship I should have returned immediately after seeing Lord Tyrawley home but was ordered by the War Office to take the care of two Highland Companies then Designed for Portugal . . .

Lord Tyrawley was extremely well when I left him and Commissioned me with his Compliments to your Lordship.

January, 1763.—With the beginning of the New Year it seems that the process of moving the troops down was continuing, and John Hunter was employed in visiting some of the camps and sending in a return of the sick and stating how many of them could be moved and how many could not. We find a return signed by him saying that at Portallegre, at Castel da Vide and at Montfort there were altogether 108 sick, of whom 24 were not in a fit state to be removed. For the removal of the fit he wanted 18 carts. This would mean that each cart could accommodate some six patients, and the journey to the nearest port on the Tagus would be about thirty miles.

Such journeys must have been very trying for the sick men, and the Director is very concerned about it. On Jan. 8, 1763, he writes from Santarem to the Adjutant-General:—

SIR,

You will be pleased to show the enclosed Return to his Excellency. I have had none very lately from Lisbon but I imagine they have about 50 patients there.

The Inconveniency I have before mentioned of the sick men's suffering from being mov'd from Place to Place continues. Mr. Tomkins writes me word, two days ago, that fourteen dyed in their Passage from the Army to Abrantes. Frederick's Regiment are remarkably sickly, many die in the hospital and many out.

Evidence comes in now that the process of clearing up was going on steadily. Lord Loudoun asked the Director to send him in details of the expenses connected with the Hospital. Mr. Young replies on Jan. 8 as follows:—

I have collected and placed in View the several articles of Expence incurred by the Hospital wherever dispersed which I hope will give you ample Satisfaction to the Question relative to what is to be charged to the King of Portugal. I have not herein included the money expended by Mr. Hunter because I have not call'd in the Account, but as He has the Honour to be near your Lordship's Person He can send in an abstract of it.

The account sent in by him amounted to £5112 os. 6d., and was presumably for the period from the opening of the hospital until the end of December, 1762. It included all sorts of details, such as the purchase of bread and meat, the hire of carpenters, and for all the sundry purchases connected with the hospital. In it, however, comes an item which is of interest as showing the amount paid to the officers of the hospital for subsistence. This amounted to £1058 7s. od., and there is a note to the effect that some of the officers had not yet taken up their allowance. There is no statement as to the amount paid to the different ranks, and

no doubt there was a difference, but if one lumps them all together it would seem that the sum per head was something over £10 a month.

In a later document the total expenses of the hospital from the opening are set out as follows :—

The following Sums of Money have been issued to Wm. Young Esq. Director of the Hospitals for the British Forces in Portugal.

1762	June	22	Cash	pd.	Mr. Young	£500
	Aug	3	Do	paid	Do	£800
	Sept	3	Do	paid	Do	£800
		25	Do	paid	Do	£800
	Nov.	29	Do	paid	Do	£800
1763	Feb.	5	Do	paid	Do	£2000
Total						£5700

Hospital Discipline.—Mention has been made previously of the measures taken to secure discipline in the hospital. The medical officers had not right of punishing the men under their charge. This was given to a combatant officer, generally a Lieutenant, who commanded the Hospital Guard.

In the Area of Lisbon.—The British Hospital Guard there was a Lieutenant, 1 sergeant, 1 corporal, and 23 privates, and there were 10 sentinels on duty day and night. Among the crimes for which the men were confined were desertion, drunkenness, and robbery, and there were two prisoners confined for 116 nights in the "Black Hole" for being concerned in murdering a Portuguese. What became of these men eventually does not transpire.

At Santarem the reports of the Provost Guard are signed by William Hodgson, Provost Martial. He seems to have believed in phonetic spelling, for one Patrick McAnalley was confined "on Suspicion of shutting a Portuguese".

The process of moving down the troops was still proceeding on Feb. 3. Francis Tomkins reports that there were only seven sick left at Montalvo. So he did not get down to Lisbon as he wished.

Hunter, however, was now at Lisbon, for on Feb. 12 he signs "An Account of the Sick in the Hospital at Lisbon", and as far as can be ascertained he remained there until he went home. It must have been while he was in Lisbon in the earlier part of the campaign that he made his observation on the hearing in fishes which he recounts in his article entitled :—

AN ACCOUNT OF THE ORGAN OF HEARING IN FISHES

In the year 1762, when I was in Portugal, I observed in a nobleman's garden, near Lisbon, a small fish-pond full of different kinds of fish. The bottom was level with the ground, the pond having been made by forming a bank all round, and had a shrubbery close to it. Whilst I lay on the bank observing the fish swimming about, I desired a gentleman who was with me to take a loaded gun and fire it from behind the shrubs. The reason for desiring him to go behind the shrubs was, that there might be not the least reflection of light. The moment the report was made the fish seemed to be all of one mind, for they vanished instantaneously, raising a cloud of mud from the bottom. In about five minutes afterwards they began to appear and were seen swimming as before.

During the month of March, the Director, Mr. Young, was busy settling up affairs at Santarem, and it seems that Hunter was in charge of the hospital at

Lisbon, for he signs all the returns, and on March 29, 1763, he writes this letter to Colonel Cosnam :—

SIR,

Mr. Maddox informs me that Lord Loudoun wanted to know what stores came last night from Santarem, the enclosed is a copy of what Mr. Young sent. I was likewise informed that my Lord Loudoun wanted to know what number of boats would be wanting to bring down ye sick and stores from Santarem as I do not know what stores there are I cannot be a judge but should suppose that four boats would bring down the sick men and their bedding and that four more would be sufficient to bring down all the rest of the stores I mean the large boats. If this is not sufficient I should imagine that their wants could be made up there. I would have waited upon His Lordship myself but I am busy preparing for the remainder of the Santarem Hospital.

The list of stores which was enclosed reads :—

15 Bails of Bedding	20 sett each
2 Do of Coverlids	60 each
1 Do of Blankets	60 each

On March 28 Lord Loudoun received a letter from Mr. Young giving him the final returns from Santarem, and in the same letter he asked leave for Dr. Morris to visit the Scaldes Baths. To this Lord Loudoun replied on March 29 :—

The arrival of the Transports makes it necessary for me to Order you to bring down the Hospital here immediately.

This makes it impossible for me to grant Dr. Morris's request which I should otherwise be glad to do.

I desire by the return of this bearer an account of what Men can join their Corps, what can be embarked in an Hospital Transport and what must be left behind, and that as far as you can in the Regimental as well as General Hospital.

Here is evidence as to the amount allowed to Regimental Surgeons for carrying medicine chests.

Robert Home Surgeon.

LISBON *March 29th. 1763.*

SIR,

You will much oblige me in applying to his Excellency the Earl of Loudoun to beg the favour of his Lordship to give an order to Mr. Bembridge, deputy Paymaster General for paying me an allowance of Baggage money for carrying the Medicine Chests of Brigadier General Burgoyne's Regiment of Light Horse which as yet I have never received, neither in England nor here.

I am not informed what allowance was in Germany but in England it is Ten pounds for a Horse and Forrage Money to the amount of seven or eight pounds more, which Mr. Tomkins, surgeon, can inform his Lordship of.

I am with Respect

Your most obedient and most humble servant
ROBT. HOME *Surgeon.*

The last return from Lisbon is on April 12, 1763, and this is a statement of the number of men from the Hospital who were not fit to be moved on to the transport, and of these there were thirty-six, and twenty-one were to go on the hospital ship.

From this we must suppose that by a date not long after April 12, the last of the Portuguese Expeditionary Force sailed for England, leaving a few unfortunate invalids behind. Not quite uncared for though, because one of the Hospital

mates, Samuel Hayes, the one who was put under arrest by Mr. Tomkins, was left behind to look after them, and as the date on which he was left is given as April 13, it is probable that the fleet sailed on that day.

The last document, which is given in full below, is the official return signed by Lord Loudoun, the Director of the Hospital, and Charles Bembridge, the Deputy Paymaster-General, certifying to the personnel of the Hospital, their rates of pay and what became of them. It is presumed, therefore, that this return was finished when the Expedition reached home.

OFFICERS OF THE HOSPITAL APPOINTED TO THE USE OF HIS MAJESTY'S FORCES
SERVING IN PORTUGAL UNDER THE COMMAND OF HIS EXCELLENCY
THE EARL OF LOUDOUN, ETC., ETC., ETC.

CHARACTERS	NAMES	FROM	TO	
Director @ £1 5 0 p.d.	William Young.	1762 Feb. 10th.	1763 May 14th.	
Physicians @ £1 0 0 p.d.	Will ^m Cadogan. Mich ^l Morris. Richard Huck.		Do. Do. Nov. 7th	{ He was appointed on Doctor Cadogan's going sick to England
	John Hunter. Francis Tomkins. Will ^m Maddox. Will ^m Young.	Feb. 10th. Do. Sept. 6th.	Do. Do. Do.	
Surgeons @ 10 sh. per diem.				
Apothecaries @ 10 sh. per diem.	Hugh Smith. Walter Hamilton.	Feb. 10th. Do.	Do. Do.	
	Moris Obryan. A. Robinson. Edm. Taylor. And ^w McDonald. Will ^m Rogerson. M. Croker. John Digby.		Do. Do. Do. Do. Do. Do.	
	Cr Nich. Jenty.	Dec. 4, 1762. Ap ^l 13th.		Died that day. { Remains in Portugal on his own affairs.
Surgeons' Mates at 5 ^s per diem.	Ter. Armstrong. Thos. Deveil. Robert Scott. David Griffith. Samuel Hayes.	Feb. 12th, 1763. Sept. 26th, 1762. May 14th. Do. Ap. 13th.		Died that day. Died that day.
	Benjamin Cluck. Edward Golding. Chris ^e Johnston. M. A. Clerck. Jnr Kingston.	Feb. 10. Do. Do. Do. Aug. 14	May 14. Do. Do. Do. Ap. 13.	Left on Duty in Portugal. Remains in Portugal on his own affairs.

I do hereby certify that the above mentioned officers of the Hospital served in the respective Qualities set down against their names.

Signed (LOUDOUN.
{ W^m YOUNG, Director.

I do hereby certify that the officers of the Hospital mentioned in the foregoing List have not received any part of their pay from me.

Signed CHARLES BEMBRIDGE.

LIST OF SERVANTS ETC., BELONGING TO THE HOSPITAL

Matron.	Mrs. Sullivan.	2	6
Quat Master.	Owen O'Neil.	2	6
Assistant Storekeeper	H. Andrews.	1	0
Head Nurses	Mary Fenton.	1	0
	Ann Milross.	1	0
	Bridget Lawson.	1	0
Cooks.	Eliz th Withers.	1	0
	Margt Cole.	1	0
	Eliz th Aldridge.	1	0
Washerwomen.	Eliz th Evans.	1	0
	Sarah Smith.	1	0
	Jane Gillan.	1	0
	Mary Armstrong.	0	6
	Mary Craufurd.	0	6
	Else Scambler.	0	6
	Cath. Morgan.	0	6
	Mary Cassady.	0	6
	Margt Barelk.	0	6
	Anne Hawkins.	0	6
	Mary Tovey.	0	6
Nurses.	Cath. Sinclair.	0	6
	Jane Murra.	0	6
	Sarah Smith.	0	6
	Jonas William.	0	6
	Anne Morgan.	0	6
	Eliz. Armstrong.	0	6
	Anne Powell.	0	6
	Thomason Redav.	0	6
	Honor Burke.	0	6
	Jane Torrence.	0	6

This concludes the account of the Expedition to Portugal and closes a chapter in Hunter's life. Peachey says that soon after his return to England Hunter made his way to the house of Robert Home, who was one of his colleagues in the campaign, and we know that later he married his daughter.

Here we may leave Hunter to pursue that work which has been, and still is, an incentive to effort and research.

CARCINOMA OF THE MALE URETHRA, WITH REPORT OF A CASE

BY HENRY MORTENSEN, MELBOURNE

CARCINOMA of the male urethra is a clinical entity of rarity, often remaining undiagnosed until the possibility of curative measures being undertaken has passed. This report of a further case is therefore of interest, and serves to demonstrate an instructive and unique pathological specimen.

Although other portions of the genito-urinary tract become frequently the seat of tumour formation, the urethra, particularly in the male, is singularly free. According to Gurlik, quoted by Englisch,¹ only 3 cases of carcinoma of the urethra were discovered in a series of 4000 tumours of the male genito-urinary tract. Since the report of a case by Kirwin in 1932,² who was able to find accounts of 98 cases in the literature, 9 other cases have been reported by Geisler,³ Beck,⁴ Lower,⁵ Boggon,⁶ Lazarus,⁷ Astraldi,⁸ and McNally.⁹ These, with the case herewith published, bring the total up to 109. The rarity of the condition, coupled with the inaccessibility of the common site for its occurrence, viz., the bulbous urethra, render early diagnosis extremely difficult, to which is also contributed the fact that the lesion is almost invariably grafted on to some more chronic and more common lesion in this situation.

CASE REPORT

HISTORY.—C. R., aged 64, was first admitted to hospital on June 6, 1935. For twenty-six years past the patient had been dilated at intervals for urethral stricture. He had had several attacks of acute retention of urine, the last occasion being six months previously. Since then he had no difficulty in micturition until seven days prior to admission, when he complained of difficulty in starting and dribbling after micturition, culminating in an attack of retention forty-eight hours prior to admission. He complained of no symptoms referable to any other system. The patient was catheterized and referred to the out-patient department for dilatation.

On July 30 he was re-admitted, complaining of severe dysuria since the passage of a sound a few days previously. On examination he presented a tense, tender mass in the perineum over the bulb of the urethra. A diagnosis of peri-urethral abscess was made.

OPERATION.—The abscess was incised and a quantity of pus evacuated. Owing to the amount of superficial infection and the general condition of the patient, it was again found necessary to explore the region, and on August 28 further pockets of pus were opened up in the peri-urethral region, and a suprapubic cystostomy performed. The condition remained



FIG. 429.—Photograph of perineum, showing the fungating mass extending from the scrotum to the anus.

unsatisfactory, the patient complaining of a great amount of pain which the incisions did not relieve, and the induration was seen to be spreading to the inguinal and scrotal regions. Two further incisions were made, and on each occasion pus was obtained, and what appeared to be typical inflammatory indurated tissue was broken down with the finger. It then became



FIG. 430.—Photograph of the specimen before sectioning, demonstrating the extent of superficial involvement and the degree of fungation.

obvious that the 'granulation tissue' was increasing rapidly in extent, and taking a definitely fungating appearance (Fig. 429). On September 28 a portion of the tissue was removed for biopsy, and revealed a squamous-celled carcinoma. The patient died fourteen days later.



FIG. 431.—Section of the upper end of the tumour in the prostatic urethra, including the ejaculatory duct. The prostatic tissue is atrophied, the epithelium stratified and papillated. The tumour mass is elevated from the surface. The tissues in the square are shown in greater detail in Fig. 432. ($\times 8$.)

PATHOLOGICAL REPORT.—The specimen was obtained by excising *en bloc* the perineum, a portion of the bony pelvis, the penis, scrotum, bladder, prostate, and rectum. Fig. 430



FIG. 432.—Higher-power view of rectangle outlined in *Fig. 431*. The remains of stratified epithelium are shown. The submucous tissues are infiltrated with strands and columns of epithelial cells showing some keratinization, with well-marked stroma reaction and numerous small round cells. ($\times 60$.)



FIG. 433.—Section of the lining of the urethra showing arrangement of the tumour cells in both stratified and simplex forms. ($\times 160$.)

demonstrates the amount of superficial involvement of the perineum and scrotum and the degree of fungation. The specimen was then split in a sagittal direction. Examination of this section reveals the ischio-rectal fossæ to be filled with neoplastic tissue. This extends, superficially, from the postero-inferior aspect of the scrotum to the anus. The neoplasm involves the anterior half of the urethra in approximately one-half of its extent, and extends proximally along it, and replacing it, to the level of the verumontanum, where it abuts on the symphysis pubis and rectal wall, without apparently infiltrating either structure. From the site of the urethra the tumour mass is traversed by multiple sinus tracts, giving the superficial portion of the growth an ulcerated appearance, the base and edge of which are extremely friable. The bladder is small and contracted, the mucosa being thick and smooth. The anal canal and rectum appear normal. The pelvic lymph-glands show no carcinomatous infiltration. There were no palpable inguinal lymph-glands.

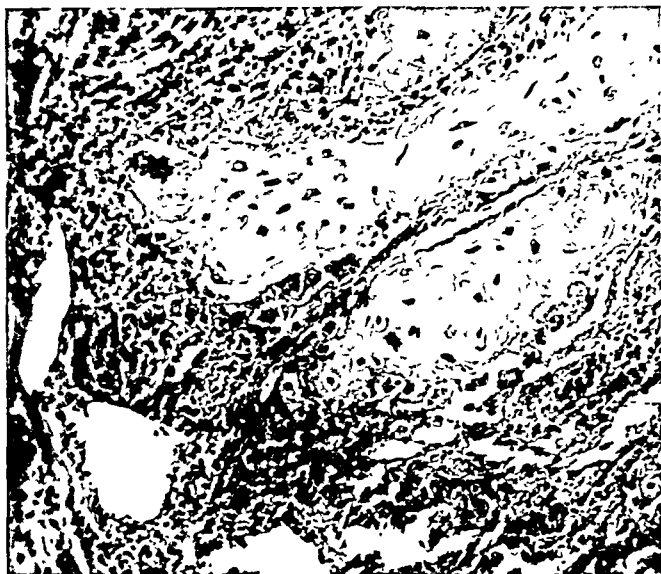


FIG. 434.—Section of carcinoma infiltrating the scrotal wall, showing marked keratinization. ($\times 290$)

MICROSCOPICAL APPEARANCES.—Microscopical specimens show a keratinizing, epidermoid carcinoma, infiltrating the urethra (*Figs. 431-433*), prostate; corpora cavernosa, and scrotum (*Fig. 434*). The pubic bones and the rectal and anal wall are free of infiltration, as are the pelvic lymph-glands.

DISCUSSION

Histologically, carcinoma of the urethra has to be carefully distinguished from that arising in the penis, prostate, or Cowper's gland, and from secondary involvement of the urethra from these regions. A large percentage of the urethral carcinomata are of the squamous-celled type, the occurrence of which, in a site normally lined by transitional epithelium, is explained by the process of epithelial metaplasia. The frequent occurrence of the pathological changes in epithelium leading to the development of leucoplakia, discovered in routine urethroscopy for the diagnosis and treatment of chronic urethral infections, stresses the importance of this factor. The papillary type of tumour is found less commonly, often in the younger class of patient, when it is of more rapid growth and of a greater degree of malignancy. A true adenocarcinoma has been described on only two occasions,

by Lower,⁵ and Olivier and Clunet,¹⁰ and arises from the glandular structures of the urethra. The tumour spreads locally by direct extension. In the anterior urethra it may creep along the urethral surface to pout at the urethral meatus, and, involving the body of the penis, cause deformation in the erect organ and a palpable nodule in its course. Farther posteriorly the invasion of the peri-urethral tissues is not so obvious, and it may be only when, as in the case here reported, the ischio-rectal fossæ and the perineum are completely filled with tumour growth that the nature of the condition is recognized. Metastases to lymph-glands occur in some 50 per cent of the recorded cases. The lymphatic drainage of the urethra is to the inguinal and deep pelvic glands. In many cases death from urinary obstruction or sepsis takes place before any inguinal involvement, but the possibility of metastasis to the deeper glands must be borne in mind. It is worthy of note that in the case history here presented, in the presence of gross local disease there was no demonstrable lymphatic involvement.

Symptomatology.—The symptoms of carcinoma of the urethra will vary with its position. One is struck with the stereotyped history given in the cases reported as occurring in the bulbous urethra. In 1885 Paul¹¹ reported a case as follows :—

Josiah C—, aged 54, a seaman, had suffered many years from stricture (he says thirty), but has never been treated for it. He sought admission on account of a perineal abscess. Under ether the abscess was opened, the stricture divided, and a full-sized gum-elastic catheter tied in. On the third day this catheter came out, and was not re-introduced, a bougie being passed daily instead. At the end of two months he was discharged nearly well, but warned to attend as an out-patient to get the bougie passed. He was an extremely nervous man, and, once out of the hospital, had not the pluck to continue treatment, the result being that in two months he had another urinary abscess worse than the first. He kept away from the Hospital until he found himself getting in a bad way, when he returned in the following condition: The scrotum and the whole of the perineum were brawny and tender. Just behind the former was a ragged foul-smelling sore, eating deeply into the perineum along the tract of the original fistula. Its edges were hard and epitheliomatous in appearance. The disease was too far advanced to permit of any attempt at removal, and came to a fatal ending in three months. At the post-mortem the growth was found to be limited to the perineum and the neighbouring glands, involving all the parts down to the arch of the pubis and spreading freely into the scrotum. The prostate and bladder were quite free.

Paul comments on this interesting case: “. . . under the circumstances I am inclined to the opinion that the growth commenced in the urethra, and was excited by the prolonged stricture, just as simple syphilitic stricture of the œsophagus ends, sometimes, in epithelioma of that structure”.

As was realized then, carcinoma of the urethra is associated with stricture in a very large percentage of the cases reported. Robb¹² had the unique experience of excising a stricture which on section proved to be infiltrated with carcinoma. It is probably the repeated passage of sounds in the treatment that provides the etiological factor. Other sources of chronic irritation, e.g., chronic infections of the urethra and their treatment by chemical agents, have been noted in the histories, but so infrequent is the occurrence of carcinoma compared with the number of patients who undergo prolonged urethral treatments that any etiological relationship must be questioned.

The earliest symptoms in this region are, therefore, obstructive in nature: difficulty in urination, and progressive diminution of the calibre of the stream.

Suspicion may be aroused as to the nature of the obstruction by the lack of satisfactory response of the stricture to dilatation, by a persistent bloody discharge associated with, or independent of, treatment, or by a thickening or induration at the site of the lesion. Subsequently a peri-urethral abscess develops, with perhaps an extravasation of urine. The pathological nature of the stricture becomes obvious when surgical treatment of the abscess does not lead to the relief of pain, or to subsidence of the induration or œdema, or when fungation leads to biopsy and the correct diagnosis. More anteriorly in the penile urethra, the recognition of the presence of a carcinoma may be relatively early, and precede the onset of obstruction. The passage of a few drops of blood at the beginning of micturition, or the presence of a bloody discharge, leads to investigation with a urethroscope, or the tumour may become visible at the meatus or palpable along the penis.

Treatment.—For the cases not hopelessly inoperable when first recognized many procedures have been adopted. In the small localized tumour in the anterior urethra partial amputation of the penis will be indicated. In two of the three cases reported by Lower, resection of the growth with end-to-end anastomosis of the urethra was performed, with striking results. One case was alive and well eight years, and the other nine years, after operation. In the third case, pathologically an extensive involvement of the urethra with adenocarcinoma, and metastasis in the inguinal glands, an amputation of the penis and glands was performed with a block dissection of the glands. This patient was well three and a half years after operation. In the case reported by Scholl and Braasch¹³ resection of the tumour was performed, and restoration of the urethra achieved by the use of a portion of the saphenous vein. This was followed by irradiation to the perineum, and a five-year cure. Huggins and Curtis¹⁴ have discussed the technique of perineal extirpation of the growth, whilst the use of radium or radiation may be considered. Should there be no palpable inguinal glands no surgery in that region is indicated. With gland involvement the prognosis is rendered graver, and, if the urethral tumour permits of removal, the glands are best treated by block dissection.

Prognosis.—In most cases the results of treatment are very poor, and by far the greatest number of cases reported have died shortly after diagnosis, either with or without surgical intervention. The possibility of surgical treatment depends on early recognition. It may be that the condition is more common than the literature would lead one to believe, and the possibility of the occurrence of urethral carcinoma should be borne in mind in any case of stricture demanding prolonged dilatation.

My thanks are due to Mr. R. D. Wright, of the Department of Pathology, University of Melbourne, for the pathological report and the micro-photographs; and to Mr. Leo Doyle, Surgeon to St. Vincent's Hospital, Melbourne, under whose care the case was admitted, for permission to publish the details.

REFERENCES

- ¹ ENGLISH, J., "Das Epitheliom der männlichen Harnröhre", *Folia Urol.*, 1907, i, 38-76.
- ² KIRWIN, T. J., "Primary Epithelioma of the Urethra", *Jour. of Urol.*, 1932, xxvii, 539-560.
- ³ GEISLER, W., "Ueber einen sehr enreifen Harnröhrenkrebs der Pars prostatica mit Impfmestastasen in der Fossa navicularis", *Arch. f. pathol. Anat.*, 1931, cclxxxi, 88-97.

- ⁴ BECK, M., "Ueber einen Fall von primärem Krebs der männlichen Harnröhre", *Zeits. f. urol. Chir.*, 1932, xxxiv, 179-185.
- ⁵ LOWER, W. E., "Treatment of Malignant Growths of the Male Urethra", *Clinical Rep., Trans. Amer. Assoc. G.-U. Surg.*, 1931, xxiv, 249-252.
- ⁶ BOGGON, R. H., "Carcinoma of the Male Urethra", *Brit. Jour. Surg.*, 1933, xx, 640-644.
- ⁷ LAZARUS, J. A., "Primary Carcinoma of the Male Urethra. Case Report", *Jour. of Urol.*, 1934, xxxi, 823-831.
- ⁸ ASTRALDI, A., "Primary Carcinoma of the Penile Urethra", *Urol. and Cutan. Rev.*, 1935, xxxix, 20-21.
- ⁹ McNALLY, A., "Primary Carcinoma of the Urethra. Report of a Case Occurring in the Male", *Jour. of Urol.*, 1935, xxxiv, 389-390.
- ¹⁰ OLIVIER and CLUNET, "Epithelioma primitif de l'Urèthre d'Origine glandulaire", *Bull. et Mém. Soc. Anat. de Paris*, 1907, lxxxii, 735.
- ¹¹ PAUL, "Old Stricture, Perineal Abscess, Urinary Fistula becoming Epitheliomatous. Case Report", *Lancet*, 1885, i, 1127.
- ¹² ROBB, J. J., "Cancer of the Male Urethra: Two Cases, with a Short Survey of the Subject", *Brit. Jour. Surg.*, 1928, xv, 605.
- ¹³ SCHOLL, A. J., jun., and BRAASCH, W. F., "Primary Tumors of the Urethra", *Ann. of Surg.*, 1922, lxxvi, 246-259.
- ¹⁴ HUGGINS, C. B., and CURTIS, G. M., "Cancer of the Male Urethra with Technique of Penis Extirpation", *Surg., Gynecol. and Obst.*, 1929, xlviii, 544.

FAT EMBOLISM: REPORT OF A CASE, WITH REVIEW OF THE LITERATURE

By A. J. WATSON, LONDON

ALTHOUGH there is much to be found in the American and Continental literature on the subject of fat embolism, there is still but scanty reference to the condition in the current text-books of surgery and orthopædics.

The following case, admitted to the Surgical Unit of the British Post-graduate School at the Hammersmith Hospital, L.C.C., was thought worthy of description as it presents so many of the classical symptoms and clinical features of the condition.

CASE REPORT

Patient L. P., male, aged 22, a printer by trade. He was admitted on April 18, 1935, to the Hammersmith Hospital, having been knocked over by a six-wheel motor truck a few minutes previously. He had sustained a compound fracture of the right tibia at the junction of the middle and lower thirds. No other injuries were found, nor was there any history of a period of unconsciousness following the accident. Memory of the accident was complete.

ON ADMISSION.—The patient was noticed to be considerably shocked, far more so than the fracture could account for by itself. Temperature, 97°; pulse, 98; respiration, 20. Operation was delayed for two hours until his general condition had improved.

OPERATION.—Gas-oxygen and ether anaesthesia. There was some difficulty with the anaesthetic, and the patient had a poor colour throughout the operation. A considerable quantity of ether was necessary to keep him anaesthetized.

The leg was first cleaned with ether soap and was then shaved. There was a lacerated wound in the lower third of the leg, and the upper fragment of the tibia was protruding through it. The wound was excised, clot removed, and the fracture reduced. There was little comminution of the fragments. The wound was packed with flavine gauze and a plaster cast applied, leaving a window over the wound. No tourniquet was used. Some liquid fat was seen at the time of operation around the fracture, but not more than would normally be expected.

SUBSEQUENT PROGRESS.—

12 Hours.—On the following morning, the patient was quite comfortable and the general condition was good. He had a slight cough with a little 'rusty' sputum. This was attributed to the difficulty in the anaesthesia. The leg was very satisfactory. Temperature, 99.2°; pulse, 104; respiration, 20.

24 Hours.—In the evening, the patient complained of precordial pain. The pulse-rate had increased to 128 and the temperature to 100.4°. Respirations were rapid and the cough was still present.

36 Hours.—On the next morning, the patient had become comatose. He had been very restless all night and had become unconscious early in the morning. He was breathing rapidly and deeply, and his pulse was full and rapid. Temperature, 102.4°.

On examination: His pupils were equal. There was no spasticity or convulsive movements of the limbs. Reflexes were present, although there was an extensor response on the sound limb. Some fine crepitations were heard at the bases of both lungs.

The possibility of intracranial hæmorrhage was considered and a lumbar puncture performed. Normal C.S.F. was withdrawn, with no definite increase in pressure. The affected leg appeared to be quite satisfactory.

48 Hours.—In the evening, the condition was about the same, although the coma was rather deeper. No localizing cerebral signs had appeared.

Hours.—On the following morning, coma was very deep, with Cheyne-Stokes on. Many small petechial cutaneous hæmorrhages over the neck, arms, and upper the trunk had made their appearance.

Hours.—In the evening, the patient was deeply unconscious, with incontinence of Death occurred early on the following morning, about 80 hours after admission to

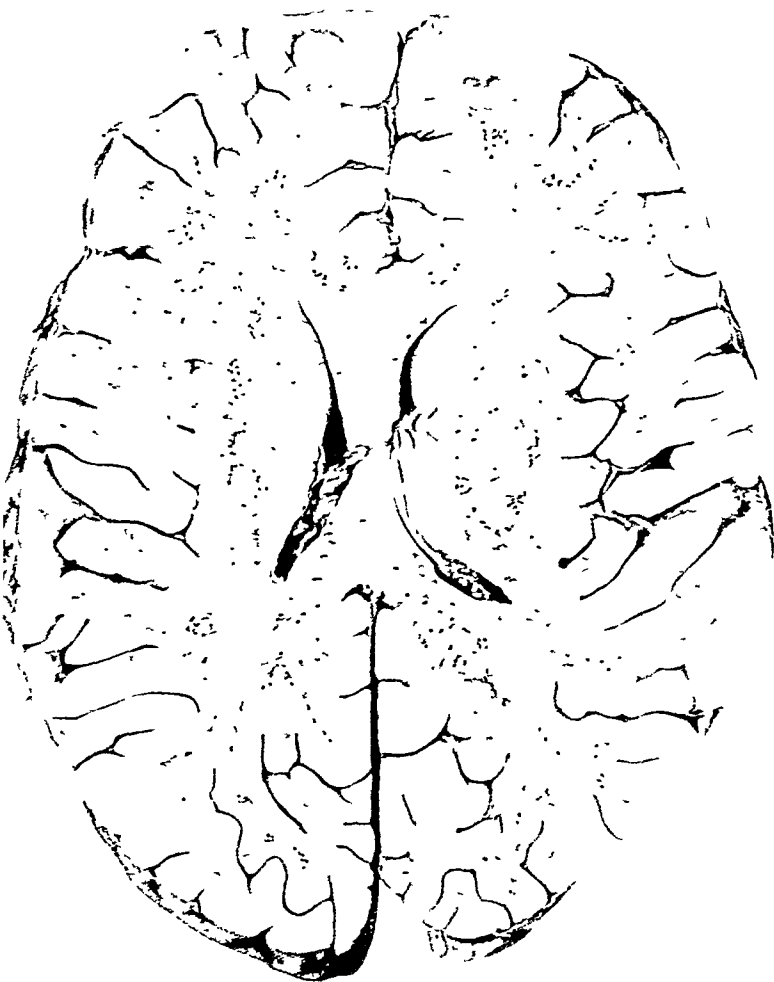


FIG. 435.—Macroscopic appearance of cross-section of brain.

he early stages, intracranial laceration or hæmorrhage was considered, but with the ent of the petechial skin hæmorrhages, fat embolism was suspected. By that condition of the patient was so critical that active treatment of any sort was thought able.

PATHOLOGICAL REPORT.—A post-mortem was carried out under the Coroner's instructions the following pathological findings:—

fracture was found to be perfectly clean with no sign of local infection. The lungs marked congestion throughout with patches of hæmorrhage beneath the visceral

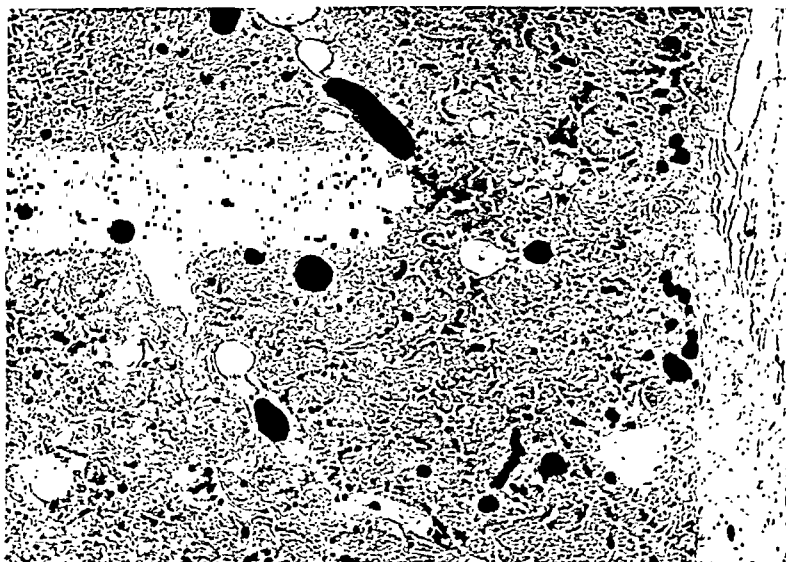


FIG. 437.—The lungs.

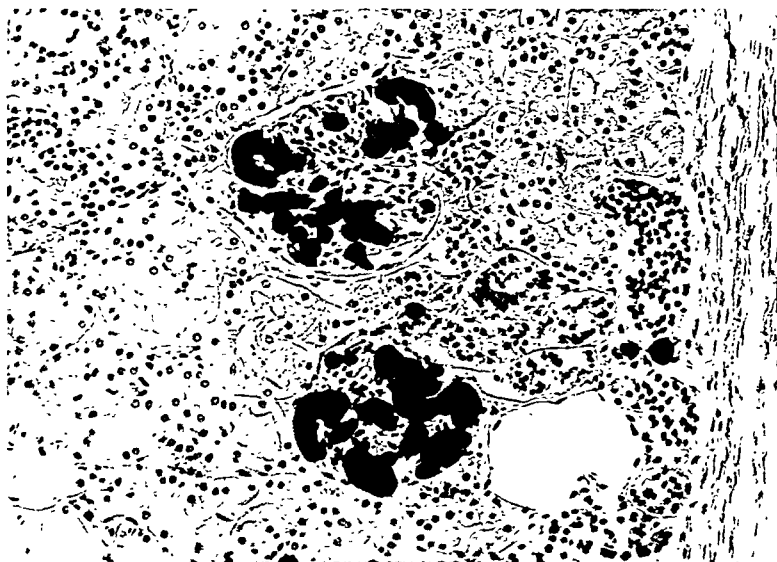


FIG. 436.—The kidneys.

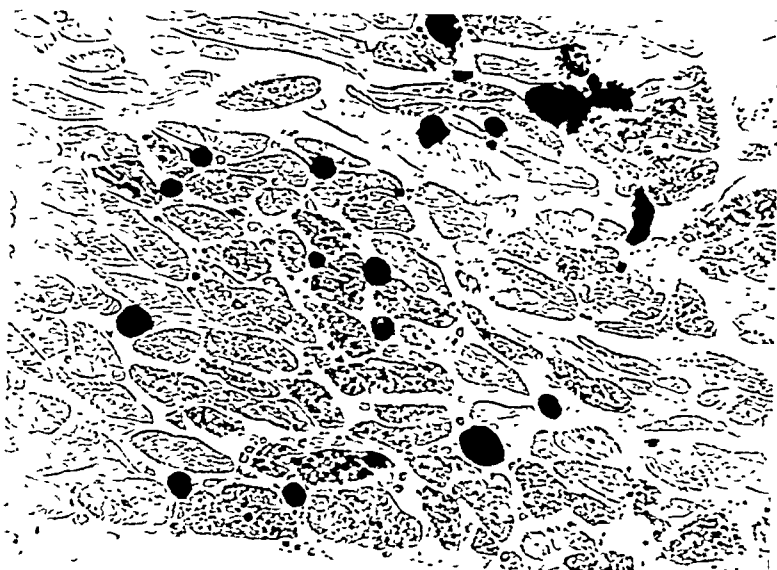


FIG. 439.—The heart.



FIG. 38.—The brain.

pleura. There was slight congestion of both kidneys. The brain (*Fig. 435*) showed many minute punctate hæmorrhages scattered through the white matter, but there was no evidence of injury to the skull or of cerebral contusion or laceration.

Microscopical Examination.—Sections of various organs stained with osmic acid showed fat embolism.

Kidneys (Fig. 436): The glomeruli contain many globules of fat, some of them elongated in the shape of the vessels. There is one globule in a tubule, but otherwise the structure of the organ is unchanged.

Lungs (Fig. 437): There is evidence of consolidation, with extensive exudate into the alveoli. Many particles of fat of various sizes are seen scattered throughout the field. Some are lying in the lumen of the alveoli, others are in the pulmonary capillaries.

Brain (Fig. 438): In this section no fat is actually visible in the small capillaries, but there is evidence of perivascular inflammatory reaction. Other sections taken demonstrated fat lying in the lumen of the vessels.

Heart (Fig. 439): Scattered through the bundles of the heart muscle are many large emboli, but there is no surrounding inflammatory reaction.

On the post-mortem findings, a diagnosis was made of acute cerebral fat embolism following a compound fracture of the tibia.

HISTORICAL

Experiments illustrating the effects of the introduction of an excess of fat into the circulation of animals were first carried out by Magendie in 1836. Magendie injected olive oil into the circulation, and found that the globules into which it split obstructed the smaller vessels and mechanically prevented the normal passage of the blood.

In 1862 Virchow injected small quantities of oil intravenously into dogs, producing dyspnoea and, in larger quantities, death. Post-mortem examination of these animals showed acute œdema of the lungs.

At about the same time Zenker first described a case of fat embolism occurring in man. His patient was a railwayman, who suffered a severe crushing injury to the abdomen, and after death large quantities of fat were seen in the pulmonary capillaries. Zenker thought that the fat had been aspirated from the stomach directly into the ruptured hepatic veins.

In 1865 Wagner described 48 cases of fat embolism occurring in man, mainly following injuries to bones and soft parts, but including in the series 12 cases associated with acute osteomyelitis, and 6 cases with chronic infections elsewhere.

Holm, in 1876, examined post mortem 21 cases of bone injury, and found evidence of fat embolism in all the cases, although they had shown no clinical signs of it whatever during life. He concluded that some degree of embolism was present in all cases of bone injury, but that clinical symptoms and signs caused by the embolism were rare.

The first complete clinical account of the condition with a review of all reported cases was written in 1880 by Scriba, who included in his article 34 cases of his own. He analysed 172 cases, and thought that in only 14 of these could death be attributed to fat embolism primarily. He found that the fat produced symptoms by mechanical obstruction of arterioles and capillaries in different parts of the body, and that this led to small areas of infarction. He considered that the cerebral emboli were of very great importance, and were the cause of death in most of the fatal cases. He recognized that the fat was excreted in the urine and that this was a point of diagnostic interest. He described what are now the classical symptoms

and clinical signs associated with the condition, but could offer no suggestions as to treatment other than the relief of symptoms.

After Scriba's complete account many isolated cases were reported, chiefly from Germany. Meeh in 1891 collected 113 fatal cases in which death had been attributed to fat embolism, but thought that only 15 of these cases really died of that condition.

Aberle in 1903 reported a number of cases following orthopædic operations, including osteotomies and manipulation of joints.

In 1913 Warthin wrote a comprehensive paper reviewing the past literature and adding some cases and observations of his own. He drew attention to the diagnostic importance of fat in the sputum, and also found emboli in the cardiac muscle, which he thought might have been responsible for some of the cardiac symptoms.

Further experimental work on the reaction of animals to the injection of fatty substances was carried out by many workers, including Lecount and Gauss, Bissell, Porter, and Lehmann and Moore—the latter throwing doubt on the source of the fat in fat embolism.

The most recent monograph on the subject was written in 1931 by Vance, who investigated 246 necropsies in his capacity of Chief Medical Officer of New York City. He found that fat embolism was commonly found at post-mortem in non-traumatic cases, but never in large quantities. Severe fat embolism was always the result of trauma received before death—a point of medico-legal importance.

A further complete historical review of the literature by Scuderi is to be found in the *International Surgical Digest* for October, 1934 (to whom the writer is indebted for many of his references.)

MECHANISM OF TRAUMATIC FAT EMBOLISM

Source of the Fat.—Blood-plasma contains fat in quantities varying with the ingestion of fatty substances. Its normal limits are from 0.2 per cent to 2 per cent, but in certain exceptional circumstances, as in diabetes, the blood fat may rise as high as 20 per cent. This type of fat is present as an emulsion of very minute particles which pass easily through the capillary network of the body and never form emboli, as they have a diameter well below that of the smallest capillary.

Fat is also present in most tissues of the body, but in some regions it is found in large quantities, forming 'depots' or reserves for use when required by the body metabolism. Depot fat is found in the subcutaneous tissue, in the omentum, around certain viscera such as the kidneys, and in the marrow cavities of long bones. It is held within the cell membranes of specialized connective-tissue cells in the form of large globules. It is the depot fat, lying in the marrow cavities of the bones, that is considered by most writers to be the source of the fat in fat embolism.

Absorption of the Fat.—In order that this fat should reach the blood-stream directly, there must be: (1) Rupture of the connective-tissue cells, liberating the fat; (2) Rupture of neighbouring blood-vessels, providing a portal of entry; and (3) Some other factor causing the free fat to pass into the circulation.

In fractures of the long bones the first two conditions are present. With the initial injury and subsequent manipulations, the tissues are lacerated and fat in the marrow-cells is liberated. The globules can often be seen floating in the blood

during operations upon bones and in compound fractures. The blood-vessels in the bone are also torn, and, as they are held within the rigid walls of the Haversian canals, they do not collapse readily, and—theoretically at any rate—remain as possible channels for the absorption of the liberated fat. It is difficult, however, to determine the precise mechanism by which the fat is made to enter the circulation.

The generally accepted explanation is that, with the formation of a hæmatoma about the fracture, there is a local rise of tension in the tissues and some of the fat is driven into the torn veins. It is also suggested that manipulation of the injured parts may force fatty globules into the circulation. Schultze demonstrated that after fracture of the tibia in dogs, no fat could be demonstrated in the femoral veins until the fracture was manipulated. Schultze and Behan suggested another factor when, by boring holes in the tibiæ of dogs, they found that the marrow cavity had a negative pressure of 20 mm. of mercury. It is possible, therefore, that the loose fat and blood may be sucked into the circulation. Unfortunately, Rothmann repeated the experiments and found that the marrow cavity was under a slightly positive pressure. Busch bored holes in the tibiæ of dogs and destroyed the marrow with a piece of fine wire. He was able to demonstrate fat emboli in the lungs of the animals, showing that fat from the traumatized marrow cavity can enter the circulation even in the absence of a fracture.

Lymphatic Absorption.—The possibility of fat being absorbed in any quantity by the lymphatics was suggested by Fritsche, who found that ligation of the veins from an injured limb did not prevent the occurrence of pulmonary fat embolism. The fat, he thought, enters the circulation via the thoracic duct.

Gauss also considered that fat was absorbed by the lymphatics, but at a slower rate than by the veins. Thus after the first appearance of fat emboli from venous absorption, a new and possibly fatal dose of fat might enter the circulation through the lymphatic trunks.

Wegelin, however, showed that olive oil injected into rabbits subcutaneously, subpleurally, and intraperitoneally, was found in the regional lymph-glands, but in a saponified state.

Busch injected olive oil stained with vermilion into the medullary cavity of long bones and found a quantity of the stained oil in the lung capillaries, but none in the lymphatic vessels draining the area and only a small trace in the lymphatic glands.

At the present time it is generally considered that lymphatic absorption plays a very unimportant part in the causation of fat embolism.

Alternative Source of Fat.—There are two factors in the pathology of fat embolism which have thrown doubt upon the hypothesis that the main source of fat is the traumatized fatty tissue at the site of injury. Firstly, the occurrence of widespread embolism appears to bear no constant relation to the severity of the trauma, and therefore presumably to the amount of fatty tissue injured and to the amount of fat liberated. Fatal embolism may follow a trivial injury, whereas a severe comminuted fracture with laceration of the subcutaneous fat may not show any clinical signs of embolism at all.

Secondly, Lehmann and Moore suggest that the actual quantity of fat available at the site of injury is probably not by itself enough to produce fatal embolism, even if it were all to enter the circulation at once. These workers found experimentally that a dog well tolerated intravenous injection of cotton-seed oil at the

rate of 1.5 c.c. per kilogram of body weight without symptoms. By comparison a man of 60 kg. could tolerate 90 c.c. of the oil without producing symptoms. But the marrow cavity of an average human femur only contains about 65 c.c. of fat. Therefore, in a fracture of a femur, the whole of the marrow fat might be absorbed without producing any serious symptoms.

Similarly Warthin states that 210 g. of fat are necessary to produce fatal embolism in man, and that this is an amount far in excess of any fat that might be liberated around a fracture. It is possible, of course, that quantitative estimation of this sort based upon experimental work in animals with fatty oils instead of natural fats may give entirely misleading results and that a much smaller quantity of fat may produce symptoms if it finds its way to vital centres.

Further experiments by Lehmann and Moore, however, suggest the possibility of an additional source of fat, namely the blood-plasma.

As stated above, the fat in the plasma is present as a very finely divided suspension or emulsion. Emulsions are unstable and can be broken up by the addition of certain agents which lead to an aggregation of these particles to form larger particles. If this occurred to the fatty emulsion in the blood-stream, the larger particles might be big enough to produce embolism.

Lehmann and Moore at first suggested that blood fat might be broken up by the products of tissue destruction circulating in the blood after an injury. They were able to demonstrate that extracts of necrotic muscle and histamine, when added to fine emulsions of cotton-seed oil *in vitro*, produced a break-up of the emulsion—the change occurring more rapidly in the coarser emulsions. These substances, however, had little effect upon the fat in the blood-serum obtained from a dog after a fatty meal.

On the other hand, fat solvents such as ether and alcohol rapidly cleared all the solutions, including the blood-serum.

Some experiments on living dogs showed that fat emboli could not be produced by the injection of histamine or of tissue extracts, but that they were found in large numbers in the lungs after the injection or even inhalation of ether following a fatty meal. The ether in the plasma presumably dissolves the fat, but when the blood reaches the lungs the ether evaporates, throwing down the fat in the form of emboli. It is a possibility, therefore, that in patients subjected to ether anaesthesia shortly after the ingestion of fat, some of the blood fat may be precipitated in the lungs, causing pulmonary fat embolism. There is as yet no clinical confirmation of this hypothesis, although it will be noticed that in the case reported here a larger quantity of ether than usual was used. No history of a recently eaten fatty meal was recorded.

Distribution of the Fat.—It appears that whatever may be the means by which the fat enters the circulation, it is found first in the veins and so passes on to the right side of the heart. When the fat mixes with the blood in the heart, the viscosity of the blood is increased, and this interferes with normal contractions. A foamy clot similar to the frothy blood in air embolism has been found in the hearts of many fatal cases. From the heart the fat is driven into the pulmonary circulation, where small emboli lodge in the lung capillaries. Local congestion and oedema follow, and small areas of inflammation are found. The result is firstly to diminish the area of lung available for pulmonary ventilation, with resulting cyanosis and dyspnoea, and secondly to produce back-pressure dilatation of the right side

of the heart. This respiratory and circulatory embarrassment may be mild in character or may prove fatal at an early stage.

Usually at a later stage emboli are found in the systemic circulation. According to Gauss, they may reach the systemic circulation by being driven through the pulmonary capillaries, or may pass from the right side of the heart to the left through the foramen ovale, which is patent in about 33·3 per cent of people. Frauendorfer suggested that the increased pressure in the pulmonary circulation causes a rise in tension on the right side of the heart, and fat and blood are forced through a foramen ovale which may not be patent under normal conditions. In the systemic circulation the fat is carried to all parts of the body and may be found in the capillaries of almost all tissues. In most of the tissues the small emboli do not produce any important changes, but in the brain very definite changes may follow. In the grey matter little damage is done as the capillary anastomosis is free, but in the white matter the arteries are end arteries and emboli produce local infarction with small areas of necrosis. This is followed by lymphocytic invasion and fibrosis. These changes in the brain produce the striking cerebral syndrome which, according to Scriba, is present in most of the fatal cases, and is well illustrated in the case here reported.

Fate of the Fat.—The fat may be excreted by the kidneys without actual damage to the renal tissues. This can be observed clinically, as fat can be readily demonstrated in the urine of most of these patients, while renal failure is unknown. Apart from this method of elimination, some of the fat is utilized by the liver, and some is removed from the tissues by phagocytic activity.

ETIOLOGY

The etiological factors in the production of fat embolism may be grouped as follows (Vance):—

Traumatic.—

- A. To bones: (1) Fractures; (2) Jarring of bones; (3) Orthopædic operations.
- B. To soft parts: (1) Injury to subcutaneous fat; (2) Injury to fatty viscera; (3) Burns.

Non-Traumatic (dubious).—Diabetes, certain poisons, childbirth, injection of oily substances.

Traumatic.—Of the traumatic cases by far the greatest number have been reported following fractures of the long bones. The site of fracture and degree of comminution, as mentioned above, do not appear to have any constant relation to the production of the condition, although there is definitely a larger proportion of cases occurring in fracture of the lower limb.

Jarring of the bones without fracture has been described as producing fat embolism. Ziemke referred to an instance following a fall from a height on to an amputation stump; Ribbert to a case where a bruise of the hip was the only visible sign of injury.

Many cases have been reported following open operations on bones, such as osteotomies and excision of joints. But it is interesting to find this complication following the manipulation of joints. Clarke described three fatal cases following the manipulation of old contracted rheumatoid joints, in which death resulted within a few hours of the manipulations. Extensive pulmonary fat embolism was found at autopsy.

Injury to the fatty subcutaneous tissue is rarely a cause of fat embolism, probably because the veins in these regions collapse and thrombose early, thus preventing absorption. Buerger reports a case, however, where fatal embolism was found in a woman who had been severely beaten by a club and had a large subcutaneous hæmatoma.

Some cases of burns show fat embolism at post-mortem. Olbrycht claimed that it occurs in second-degree burns when the subcutaneous fat is liquefied and the smaller vessels are opened. Most investigators, however, agree that in burns fat embolism is never more than a minor associated condition.

Non-Traumatic.—In diabetes, lipaemia may be seen. It is possible that the fine particles of fat seen in the blood-serum may coalesce to form droplets large enough to produce emboli. Fischer, Sandby, and Barling have described cases of this kind. Graham did not consider that the emboli in these cases were derived from the fat in the blood.

Groendahl described fat embolism following poisoning by phosphorus, potassium chlorate, potassium cyanide, phenol, alcohol, and various other agents; but did not prove the etiological relationship. Warthin reported a case following childbirth where probably injury to the pelvic fatty tissue was responsible.

Fat embolism has been found following injections of arsphenamine (Burns and Bromberg), but whether the drug or the trauma associated with the injection produced the emboli it is difficult to say.

Carr and Johnson described an interesting case of fatal embolism four hours after the injection of 50 c.c. of sterile cotton-seed oil into the urethra to facilitate the passage of a bougie through a stricture. The embolism was of the pulmonary type.

Macias de Torres noted syncopal attacks with hæmoptysis following the injection of lipiodol into the Fallopian tube, which he attributed to fat embolism.

Summarizing the etiological factors producing fat embolism, Vance stated that in from 12 to 14 per cent of all types of cases of non-traumatic death, a slight degree of fat embolism might be seen. In 59 cases of fatal fracture of the lower limb, 80 per cent showed very slight or no embolism, while in the remaining 20 per cent serious embolism could be demonstrated.

The conclusions drawn are that a slight degree of fat embolism may be found at autopsy in almost any condition and is of little significance; whereas a severe degree is indicative of injury and might be considered in some cases to be the cause of death.

SEX AND AGE INCIDENCE

There is no apparent sex differentiation except in so far as men are more liable to severe bone injuries than women. It is certain, however, that fat embolism is very rare in children. According to Landois, the usual age incidence is between 20 and 50, when the fat in the bones contains the highest proportion of olein, which is more fluid at body temperature than is either palmitin or stearin. Children's bones, on the other hand, contain relatively smaller amounts of fat, which is mainly composed of palmitin and stearin.

CLINICAL TYPES

There are two main clinical types of this condition : the pulmonic, where the symptoms are due to massive embolism in the lungs ; and the systemic or cerebral, where the emboli are scattered through the body but produce symptoms by obstructing the cerebral vessels. Warthin also suggested a third cardiac type, where the symptoms of cardiac failure might be primarily due to embolism of the capillary terminations of the coronary vessels of the heart.

All cases fall into one or other, and sometimes both, of the two main types described above.

SYMPTOMS AND SIGNS

Pulmonary Fat Embolism.—Groendahl distinguished two clinical varieties of pulmonary fat embolism. One occurs suddenly within a few hours of injury when presumably the circulation is suddenly flooded with fat ; the other and commoner variety has a more gradual onset, usually about the third or fourth day after the accident. The earliest signs are generally slight restlessness, with some increase in the pulse-rate and temperature. In a compound fracture this is often mistaken for local infection, but there is no increase in local pain nor sign of inflammation at the site of injury.

Accompanying the fever there is dyspnoea, which later may develop into definite air-hunger. Cyanosis is present when the capillary bed of the lung is grossly obstructed. Precordial pain may be an early symptom, associated with a feeling of constriction around the chest. A cough develops, and frothy sputum may be coughed up with flakes of blood streaking it. True 'rusty' sputum is uncommon. In the chest signs of congestion appear, and moist râles are audible over the whole of the lungs. Dilatation of the right heart and signs of cardiac failure may be present later. Hyperpyrexia is said by some to be a characteristic feature of the condition, but it probably does not occur until the emboli reach the heat-regulating centre in the brain. Usually the temperature remains about 102° – 103° .

In the acute variety of pulmonary embolism the signs are those of an acute oedema of the lungs, which may prove rapidly fatal. In the slowly developing type the signs resemble much more closely bronchopneumonia, and doubtless many mild cases treated as such recover without the diagnosis of fat embolism being made.

Systemic Fat Embolism.—As the emboli pass through the pulmonary capillaries into the systemic circulation, embolic phenomena appear elsewhere, notably in the central nervous system. Although the pulmonary signs appear first as a rule, sometimes cerebral irritation is present from the beginning, and may be the first noticeable symptom. Usually the two types develop more or less concurrently. Restlessness and excitement are the usual prodromal signs, and with the spread of the emboli to the brain and cord delirium usually follows.

According to Gauss, at first a generalized irritation of the nervous tissue is produced, and this leads to confusion of psychosensory and psychomotor stimuli, which appears clinically as delirium. After the initial irritation the emboli cut off the blood-supply to the nerve-cells, resulting in their necrosis and death. At this stage the delirium passes into coma, which becomes deeper and deeper, finally ending in death if the vital centres become involved.

The clinical features in the early stages resemble delirium tremens very closely, which probably accounts for that condition being considered in many text-books as a not infrequent complication of fracture. The delirium tends to pass slowly into stupor and coma.

With the onset of coma, various neurological signs appear. Cortical irritation may be present, producing muscular rigidity and clonic convulsions. Paralysis of muscle groups, pupil changes, and loss of reflexes follow. Finally, the coma becomes deeper, and incontinence of urine, Cheyne-Stokes respiration, and a failing pulse precede the fatal termination. Cerebrospinal fluid is usually normal, although there may be an initial increase in pressure. Blood-pressure falls with the failing circulation.

Signs of localized cerebral compression are usually absent, but the diagnosis from intracranial hæmorrhage or cerebral contusion is often a matter of some difficulty.

Simultaneously with the distribution of the fat emboli through the central nervous system, emboli occur in most tissues of the body. Petechial hæmorrhages may often be seen in the skin, usually over the neck, arms, and upper part of the trunk. Emboli in the kidney occur, and fat is passed in the urine, but no sign of inflammation or other renal damage shows itself. Emboli in the liver and other viscera do not appear to produce any demonstrable symptoms or signs. Death may occur within forty-eight hours of the onset of the condition, but is usually delayed for five or six days.

PATHOLOGICAL FINDINGS

At post-mortem the following are the chief findings as described by Vance :—

Lungs.—The lungs appear cyanotic. There are many areas of congestion and the surface appears œdematous. Frothy fluid can be squeezed from the lung. There are small hæmorrhages visible beneath the visceral pleura.

Microscopical Examination (see Fig. 437).—Many emboli can be stained and are visible in the small arterioles and capillaries. Globules may be broken up in the smaller capillaries. The alveoli contain fluid and red blood-cells.

Brain (see Fig. 435).—On section through the brain, a large number of pin-point areas of hæmorrhage are seen scattered through the white matter, giving a “Cayenne-pepper” appearance to the cut surface of the organ. The grey matter of the cortex is free from any change.

Microscopical Examination (see Fig. 438).—The grey matter is not seriously affected. In the white matter pericapillary hæmorrhage is visible, and there may be an area of necrosis surrounding the vessels. Lymphocytes are also seen in the localized inflammatory zone. Fat globules are visible in the centre of the vessels.

Kidneys.—Macroscopically, there is little, if any, change to be seen in the kidneys. Occasionally, subcapsular hæmorrhage is visible.

Microscopical Examination (see Fig. 436).—Characteristically the glomeruli contain snake-like emboli of fat often occupying the whole of the glomerular capsule. The tubules are unchanged.

Heart.—Streaky hæmorrhages may be seen on the surface and in the substance of the ventricular muscle. The blood in the chambers of the heart may contain quantities of congealed fat.

Microscopical Examination (see Fig. 439).—The capillaries in the muscle contain many emboli.

DIAGNOSIS

As mentioned above, the pulmonary signs in fat embolism resemble closely those of bronchopneumonia, and the cerebral signs those of delirium tremens.

Are there any features to enable the diagnosis of fat embolism to be definitely established?

The presence of fat in the urine has been considered by some as pathognomonic. It is present in all cases of fat embolism, but may be present following injuries without there being any clinical signs of the condition. Riedel found fat in the urine of 42 per cent of cases with fractures. Thus, although large quantities of fat suggest fat embolism, it does not exclude the diagnosis of other complications.

Warthin considered that fat in the sputum was a more reliable sign. He also described the detection of fat in the retinal vessels by the ophthalmoscope. Petechial skin hæmorrhages are characteristic, and, if associated with the other clinical features, make the diagnosis reasonably certain.

Jirka and Scuderi have recently investigated the X-ray appearances of the lungs of dogs which had been given fat embolism experimentally. They have found a uniform mistiness in the fields in all cases. They have not investigated the X-ray appearances in man as yet.

To sum up, the diagnosis must be suggested by the clinical sequence following a definite injury, and the above-mentioned diagnostic points are mainly confirmatory.

Speed quotes a useful dictum with regard to the time of onset as follows: Shock, 3 hours; fat embolism, 3 days; pulmonary embolism, 3 weeks.

PROGNOSIS

In an established case with cerebral signs, the prognosis is very grave: the majority die. Where the onset is delayed and the chest signs preponderate, the prognosis is good. Most patients recover, and there are no permanent after-effects. Hyperpyrexia, a rapid pulse, and falling blood-pressure are grave prognostic signs. Generally speaking, only the severe cases are diagnosed, while the milder cases are either overlooked or thought to be suffering from something else. Therefore, the prognosis appears to be worse than it actually is.

TREATMENT

Since the distribution of fat emboli through the lungs and brain is followed rapidly by inflammatory changes in these organs, the main object of treatment has been to prevent the fat from entering the circulation. When once fat embolism has occurred it is unlikely that any form of specific therapy will be very useful, and the relief of symptoms and avoidance of cardiac failure are the main aims of treatment.

Warthin, however, recommended the injection of a solution of 2 per cent sodium carbonate intravenously, hoping that it would combine with the fat to form a soluble soap. Schanz advised the administration of large quantities of intravenous saline to wash the emboli through the capillaries. Neither of these methods have been found successful, and the latter has the objection that it might embarrass an already overloaded heart.

PREVENTION

All writers are agreed that the avoidance of unnecessary handling or rough manipulation of fractures is an important factor in the prevention of the absorption of fat from the site of fracture. This, of course, applies to the first-aid management as well as to subsequent interference. During operations upon bones and fractures the use of Esmarch's tourniquet is advised by Ryerson, who found in experiments on dogs that the degree of fat embolism was less where the tourniquet was used, especially if it was allowed to remain on after the operation, and was only gradually released. Experiments by Caldwell and Huber showed similar results, and also demonstrated that the amount of fat embolism developing after the removal of the tourniquet depended on the activity of the animals. It seems, therefore, that immobilization after operation on bones and fractures is also an important factor in prophylaxis. Warthin, in addition to the points mentioned above, advises the use of a saw rather than a chisel in orthopædic and bone operations when possible.

Drainage of the veins coming from the injured area has been advised by Reiner, who suggested the insertion of a glass cannula into the top of the saphenous vein in lower-limb injuries so that it projected into the femoral vein. The tourniquet was then released, and the blood containing the loose fat was washed out first, before the circulation in the limb was fully established.

Wilm, thinking that lymphatic absorption was an important factor, advised drainage of the thoracic duct in the neck. Venesection to remove circulating blood containing fat has been recommended by Warthin.

The use of ether anæsthesia has been referred to elsewhere in this article, and it seems possible that, where another anæsthetic can be used with safety, ether should be avoided in any case where there is gross injury to bones or fatty tissue.

CONCLUSIONS

1. Fat emboli may be demonstrated in small numbers in about 14 per cent of all post-mortems. In cases of injury, particularly in fractures, a large degree of fat embolism may be found in the lungs and in the brain. This in a small proportion of cases produces inflammatory changes which may prove fatal.

2. The fat globules producing the embolism probably come from the site of injury, although the precise mechanism of absorption is not known. The possibility of normal blood fat being a source of the emboli has been considered in the light of the experiments of Lehmann and Moore.

3. The symptoms and signs of fat embolism fall into two main groups—pulmonary and systemic. The clinical features of these groups have been here described, and a case illustrating both syndromes has been reported.

4. The diagnosis of fat embolism is made mainly on the clinical progress and by exclusion of other complications of injury. Apart from this the presence of fat in the urine and in the sputum, and the development of a petechial hæmorrhagic rash, are important diagnostic signs.

5. Treatment of the established condition is unsatisfactory, as no method so far tried has been successful in dislodging or breaking up the fatty globules in the capillaries. The main points in the prevention of the absorption of fat into the general circulation have been outlined.

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BIBLIOGRAPHY

- ABERLE, *Zeits. f. orthop. Chir.*, 1907, xix, 19.
 BUERGER, *Urtlschr. f. gericht. Med.*, 1910, xxxix, 159.
 BISSELL, *Jour. Amer. Med. Assoc.*, 1926, lxvii, 1916.
 BURNS and BROMBERG., *Amer. Jour. Syph.*, 1930, xliii, 14.
 BUSCH, *Virchow's Arch.*, 1866, xxxv, 321-388.
 CALDWELL and HUBER, *Surg. Gynecol. and Obst.*, 1917, xxv, 650.
 CARR and JOHNSON, *Jour. Amer. Med. Assoc.*, 1935, civ, 1973.
 CLARKE, *Ibid.*, 1927, cxxxviii.
 FISCHER, *Munch. med. Woch.*, 1922, lxix, 814.
 FRAUENDORFER, *Beitr. z. gericht. Med.*, 1924, 1, 6.
 FRITSCH, *Deuts. Zeits. f. Chir.*, 1910, cvii, 456.
 GROENDAHL, *Ibid.*, 1911, iii, 56.
 GRAHAM, *Jour. Med. Research*, 1907, ii, 459.
 GAUSS, *Arch. of Surg.*, 1924, ix, 593.
 HOLM, *Beitr. z. Lehre v. d. Fett-embolie*, 1876. Munchen.
 JIRKA and SCUDERI, *Ann. of Surg.*, 1936, xxxiii, 708.
 LANDOIS, *Deut. med. Woch.*, 1926, lii, 283.
 LECOUNT and GAUSS, *Trans. Chicago Path. Soc.*, 1915, ix, 251.
 LEHMANN and MOORE, *Arch. of Surg.*, 1927, xiv, 621.
 MAGENDIE, *Leçons sur les Phénomènes phys. de la Vie*, 1836, ii.
 MACIAS DE TORRES, *Rev. franç. de Gynéc. et Obst.*, 1934, xxix, 1836, 218.
 MEEH, *Beitr. z. klin. Chir.*, 1891, vii, 421.
 OLBRYCHT, *Deut. Zeits. f. d. g. gericht. Med.*, 1922, 1, 642.
 PORTER, *Boston Med. and Surg. Jour.*, 1917, 176.
 REINER, *Münch. med. Woch.*, 1907, liv, 2004.
 RIBBERT, *Deut. med. Woch.*, 1900, xxvi, 419.
 RIEDEL, *Deut. Zeits. f. Chir.*, 1887, viii, 571.
 ROTHMAN, *Munch. med. Woch.*, 1913, lx, 1664.
 RYERSON, *Jour. Amer. Med. Assoc.*, 1916, lxvii, 657.
 SAUNDBY and BARLING, *Brit. Med. Jour.*, 1882, i, 781.
 SCHANZ, *Zentralb. f. Chir.*, 1910, xxxvii.
 SCRIBA, *Deut. Zeits. f. Chir.*, 1880, xii, 118.
 SCUDERI, *Internat. Surg. Digest*, 1934, xvii, 4, 195.
 SCHULTZE, *Arch. f. klin. Chir.*, 1918-19, iii, 573.
 SCHULTZE and BEHAN, *Munch. med. Woch.*, 1912, lix, 2869.
 SPEED, *Fracture Dislocations*, 1916.
 VANCE, *Arch. of Surg.*, 1931, xxiii, 424.
 VANCE, *Amer. Jour. Surg.*, 1934, xxvi, 27.
 VIRCHOW, *Ges. Abh. zur wiss. Med.*, 1862, 726.
 WARTHIN, *Internat. Clinics*, 1913, iv, 17.
 WAGNER, *Arch. f. Heilk.*, 1862, iii.
 WEGELIN, *Schweiz. med. Woch.*, 1923, iv, 133.
 WILM, *Cor-Bl. f. schweiz. Aertzte*, 1910, xl, 197.
 ZENKER, *Beitr. z. norm. u. pathol. Anat. d. Lunge*, 1862.
 ZIEMKE, *Deut. Zeits. f. d. g. gericht. Med.*, 1922, 1, 193.

PYELOGRAPHY IN RENAL HYDATIDS

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THE intermediate form of echinococcus by its expansion cavitates the tissues in which it lies. Its natural form is spherical and its urge is to maintain this shape. Variations occur, however, through the different resistances of the tissues invaded. The cyst may extend far beyond the natural limits of the kidney, but the inner part of the circumference as a rule gains apposition with the renal pelvis or one of the calices. The papillæ undergo absorption and the characteristic structure of the

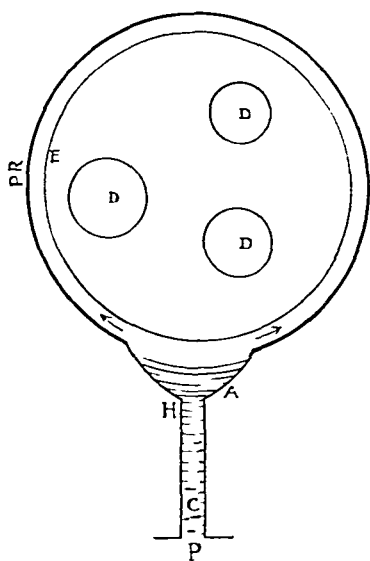


FIG. 440.—Diagram illustrating how the 'wine-glass' sign of Surraco and Mezzerar arises. A crescentic area (A) is not occupied by the parasite, and is penetrated by the opaque medium. If the ectocyst (E) also bulges into this space the shadow of the calix is cut off abruptly at the point H. Irregular quantities of the opaque fluid may penetrate between the pericyst and ectocyst in the direction of the arrows. The form of the 'wine-glass' may be clearly seen, C representing the stem. P, Renal pelvis; C, Middle calix; PR, Pericyst; D, Daughter cysts.

minor calices is destroyed, being replaced by a cup-shaped cavity into which debouches the terminal part of the major calix (Figs. 440, 441). If this bulge in the spherical outline of the cavity is occupied by a similar bulge on the part of the parasite the opaque medium used is prevented from entering, and the pyelogram shows an abrupt termination of the calix duct, like a broken pipe-stem (Figs. 442, 453). More commonly this bulge is left unoccupied, so that the medium penetrates it, giving a crescentic or cup-shaped shadow in continuity with the calix. The appearance is that of the hollow part of a champagne glass supported by the stem, the base being represented by the adjacent part of the pelvis proper (see Fig. 440).

This is the sign of the 'wine-glass' described by Surraco and Mezzera as characteristic of the condition. The animal in situ so fills the spherical part of the cavity that the fluid medium cannot enter. Sometimes, however, under pressure some of it percolates between the parasite and the cyst wall, giving rise to irregular and smudgy shadows (Figs. 447 and 459). If the pericyst or wall of the cyst is sufficiently impregnated with lime deposits it may produce in the plain X-ray plate a typical circular shadow. (Figs. 442-445, 447, 449).



FIG. 441.—Posterior half of left kidney in section. Two primary cysts are present. The upper one does not communicate with the pelvis but is pushing the upper and middle calices apart. There was a small communication between the two cysts. The section has been made exactly through this orifice, which is indicated by the upper rod. The lower rod shows the course of the middle calix. The small bulging depression which is responsible for the 'wine-glass' sign is seen covered by the lateral part of this rod. (Author's case).

Pseudo-closed Cysts.—When the echinococcus is intact and the cyst communicates with a calix, the appearances in the pyelogram are as follows:—

First Type.—The communicating calix appears somewhat drawn out and rigid. It terminates abruptly at the rim of the calcified ring, showing neither diminution in bore nor dilatation. (Fig. 442.)

Second Type.—The calix does not terminate abruptly, but ends in a crescentic or cup-shaped shadow on which the circular calcified ring rests like an acorn in its cup. (Figs. 445 and 449.)

Third Type.—In addition to the form shown in the above, flame-like areas of shadow pass out into the area bounded by the circular calcified ring and

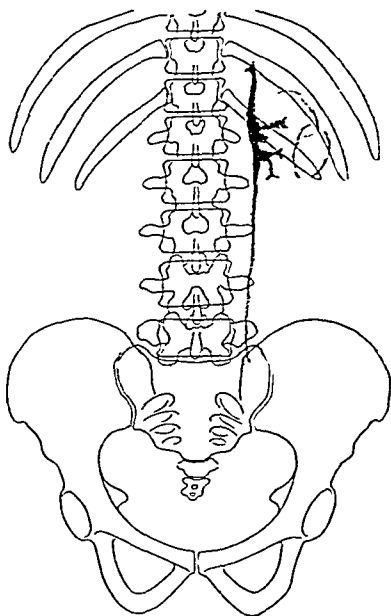


FIG. 442.—Diagram of pyelogram of the same kidney as shown in Fig. 441. The patient had no symptoms referable to the kidney and died from cancer of the lung a few weeks after this pyelogram was taken. The lower cyst shows a complete calcified ring, the upper an incomplete ring. The parasite was intact and the ectocyst extended right to the middle calix, which ends abruptly. Both upper and middle major calices are somewhat lengthened, and pressed apart by the upper cyst.

FIG. 443.—Pyelogram of the same kidney as shown in Fig. 441. The injection was made on the excised kidney. The pressure of the injected fluid has ruptured the ectocyst. The wine-glass sign is well seen. The opaque medium has passed through the middle calix and penetrated into the space within the ectocyst, clearly defining numerous daughter cysts. The fluid has also passed around the ectocyst and ruptured the ectocyst of the upper hydatid, which is also packed with daughter cysts. There was no communication between the upper calices and the cysts.



a definite circumferential ribbon of shadow may appear reinforcing the latter. (Fig. 447.)

Fourth Type.—In rare cases the flame-like or circumferential shadows may pass out directly from the abrupt termination of the calix, the crescentic area being absent. (Fig. 459.)

When the calcified ring shadow is absent the same appearances are seen except for their relation to this ring.

The above are the typical and pathognomonic characteristics of the pseudo-closed cyst. Clinically such cases cause none of the usual symptoms of renal colic, nor are they accompanied by the passage of hydatid membrane, scolices, or hooklets

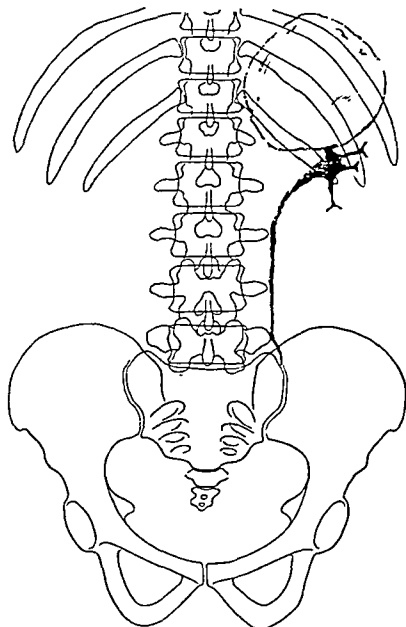


FIG. 444.—Diagram of pyelogram of large calcified cyst of the upper pole of the left kidney. The calcified circular area of the cyst is clearly seen. The wine-glass sign is suggested, but is not clearly marked, as the pyelogram was an intravenous one. The parasite was intact. (Roche and Mackenzie's case.)

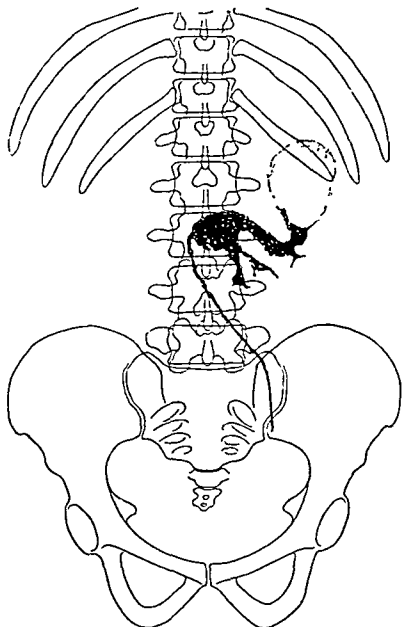


FIG. 445.—Diagram of pyelogram of pseudo-closed cyst, showing the wine-glass sign. The cyst is outlined by a ring shadow of calcification and rests on the wine-glass like an acorn on its cup. The pelvis has been pushed down and the ureter displaced medially. (After Surraco and Mezzera.)

in the urine. They are usually described as closed cysts in the literature, though in reality they are open cysts with intact parasite. They are not uncommon, and occurred in the only two personal cases I have had the opportunity of investigating. Such cases are illustrated in Figs. 442, 443, and 459.

Open Cysts.—

Pyelograms after Rupture of Ectocyst.—The echinococcus consists essentially of a completely closed membrane—the ectocyst and its contents. By this limiting laminated membrane, it maintains close apposition to the pericyst wall, partially by the tension of the contained fluid and partially by points of direct adhesion. Within this closed membrane are the hydatid fluid, the brood capsules—attached or loose—free scolices, and hooklets. At the stage when the patient is under examination there are usually daughter cysts which arise as a protective process

set in motion by the slight trauma to which well-developed parasites are inevitably subjected. The limiting membrane of the animal in about half the cases eventually ruptures at the unsupported point where the cyst cavity communicates with the calix.

Various events may follow on such a rupture. If the contents of the space within the ectocyst consist chiefly of fluid, scolices, and small daughter cysts, these are extruded into the kidney pelvis, and through the peristaltic motion of the latter are conveyed to all calices and down the ureter; or the elastic laminated membrane may contract forthwith, and even turn inside out, expelling its contents into the

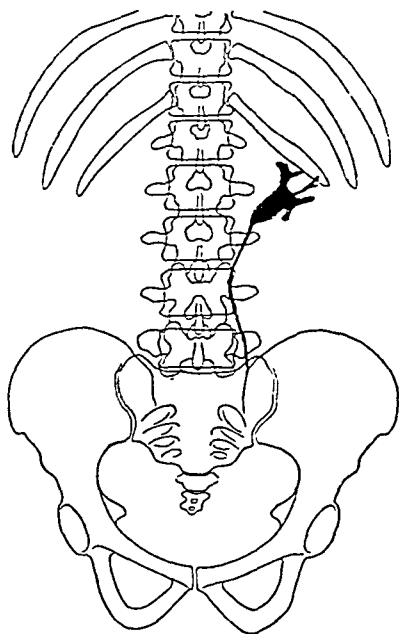


FIG. 446.—Diagram of pyelogram of cyst of the lower pole. The lower calix enters the typical crescentic space. The parasite was intact. The cyst was of the pseudo-closed variety. (After Woodman.)

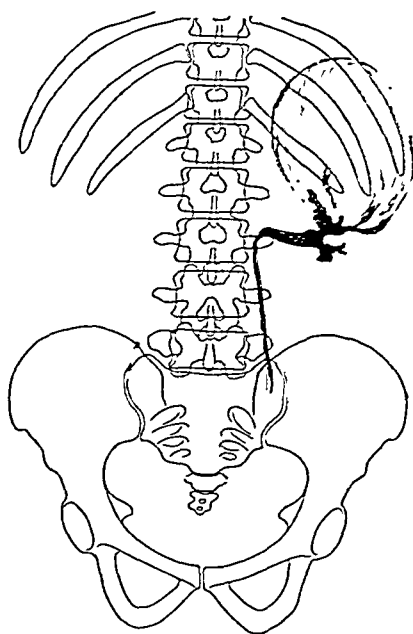


FIG. 447.—Diagram of pyelogram of cyst of the upper pole. The parasite was intact, but the opaque medium has not only filled the crescentic space at the base of the cyst but has penetrated between the pericyst and the ectocyst, outlining the circumference and showing other indefinite areas of shadow. The cyst has pressed the pelvis over so that it forms a right angle with the ureter like a wilted daffodil. (After Surrao and Mezzera.)

cavity of the cyst proper. It may then be itself expelled, or lie like a crumpled-up piece of tissue paper in the cyst cavity side by side with its own previous contents.

If, however, as frequently happens, the ectocyst surrounds a packed and almost solid mass of daughter cysts, the latter support it, and it remains roughly in apposition to the pericyst, extruding however its smaller components through the orifice caused by the rupture.

In these cases of open hydatid cyst, the pyelographic appearances are quite characteristic, especially if an adequate amount of fluid is run in, and the injection continued to the point where the patient feels the discomfort of pressure. The shadow of the crescentic bulge then becomes continuous, with a dense shadow which is well-defined and circular, and represents the cyst cavity proper (Fig. 450). In cases, however, in which the daughter cysts are numerous and closely packed,

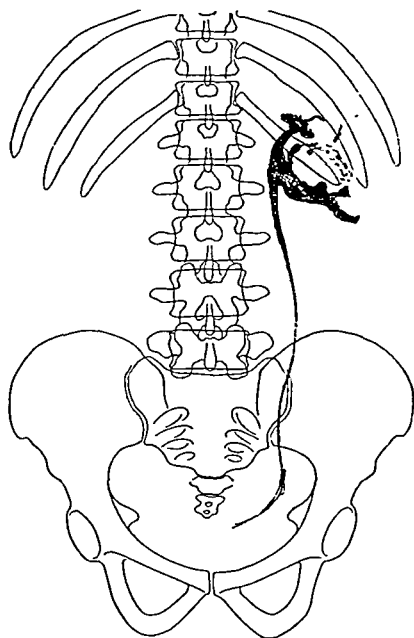


FIG. 448.—Another example of pseudo-closed cyst. The opaque medium has penetrated between the pericyst and the ectocyst. (After Surraco and Mezzera.)

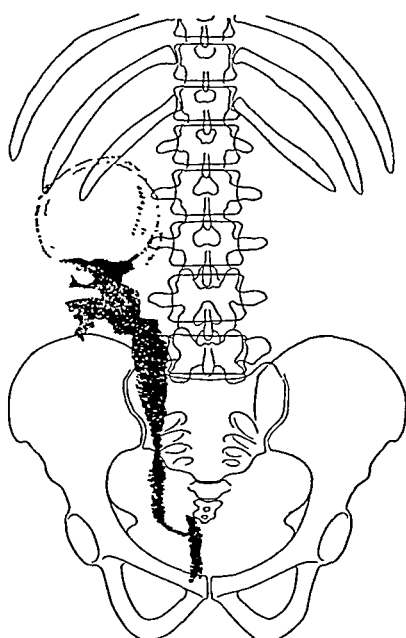


FIG. 449.—Open cyst of the upper pole, showing the wine-glass sign. The cyst was degenerated and purulent, and very little of the opaque medium has entered it. The ureter is dilated from the constant passage of hydatid material. The middle and lower minor calices show normal shadows. (After Hurley.)

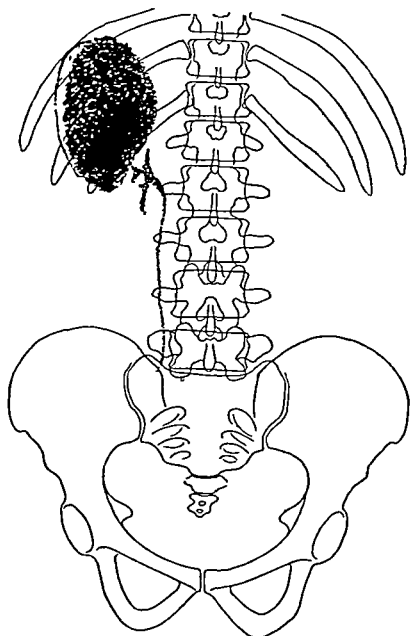


FIG. 450.—Pyelogram of open cyst of the upper pole. The daughter cysts have been pushed to the margin by the pressure of the fluid and are indicated by a mottled area. The rest of the pelvis is normal. (After Surraco and Mezzera.)

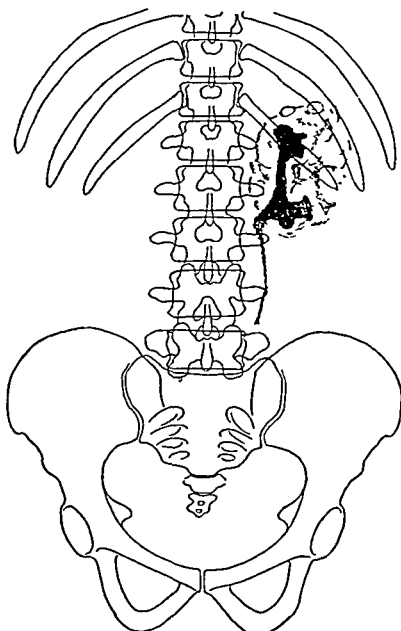


FIG. 451.—Diagram of pyelogram in open cyst. The opaque medium is seen to penetrate between the daughter cells, outlining them. (After Lee-Brown.)

very little fluid runs in except in irregular patches and more particularly around the rim (*Figs. 449 and 459*). As Surraco and Mezzera demonstrated, the mottled area may be confined to one portion of the circumference to which the pressure of the fluid forces the daughter cysts (*Fig. 450*). In fortunate cases the circular areas of the latter are well defined as filling defects throughout, as illustrated in *Figs. 443 and 451*.

The wine-glass sign is usually present. In addition there may be filling defects in the pelvis or the unaffected calices owing to the presence of escaped daughter cysts.

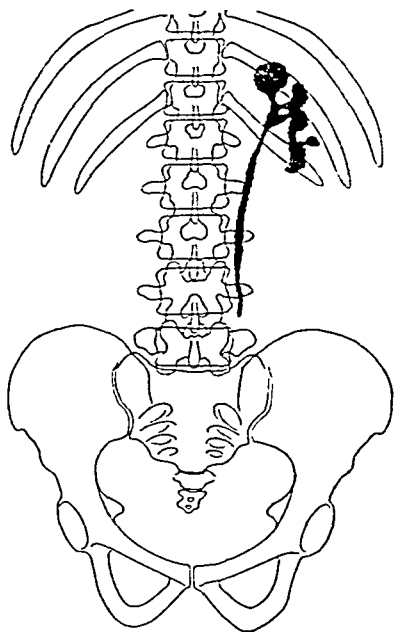


FIG. 452.—Pyelogram of a kidney with an hydatid cyst lying in front and attached to the pelvic wall. The calices are club-shaped, and there is a filling defect in the pelvis due to a urate calculus. The clubbing of the calices and the narrowing at the pelvi-ureteral junction are probably due to the calculus and not to the hydatid. (*After Vedenski.*)

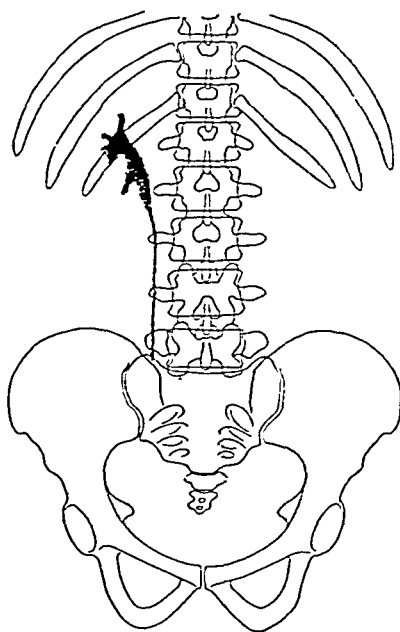


FIG. 453.—Hydatid cyst of the lower pole. The lower major calix terminates abruptly with no evidence of minor calices. The cyst was probably of the pseudo-closed variety with the ectocyst bulging into the crescentic space, obscuring the wine-glass sign. Cf. *Fig. 442*. (*After Woodman.*)

The pressure of the injected fluid may actually rupture the ectocyst, though this must be rare in the living. In one of our cases, however, this event occurred in making a pyelogram of the excised specimen. A typical picture of an open cyst was thus obtained, although the pyelogram made with the organ in situ showed a closed cyst. (*Figs. 442, 443.*)

Closed Cysts.—Hitherto we have dealt with the common variety of cysts—the open or pseudo-closed. In cases where there is no communication between the cyst and the pelvis the appearances are not so characteristic. The pyelogram is similar in many cases to that produced by a solitary cyst or even a tumour. The kidney may be displaced (*Figs. 454, 456*), complete calices may be obliterated by pressure (*Figs. 455, 457*), the ureter may be pushed inwards towards or even across the vertebral column. Sometimes a cyst may push itself into the angle between two of the major calices, separating them in such a way that they form a right angle

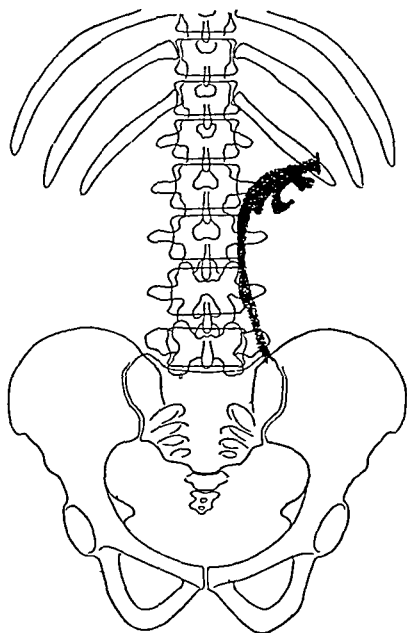


FIG. 454.—Massive closed cyst of the middle portion of the kidney, nearly bisecting it. The kidney is folded over the upper part of the cyst and the urter is pushed medially. There is some dilatation of the ureters and clubbing of the calices. (After Racić.)

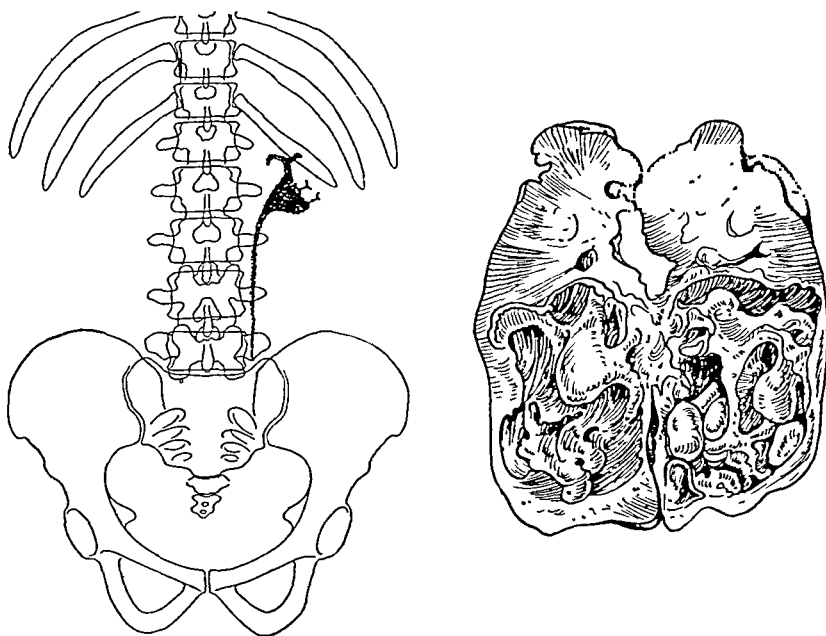


FIG. 455.—Closed cyst of the lower pole which has completely obliterated the lower calix. The remainder of the pelvis is normal. The figure on the right shows the excised kidney. (After Craig and Lee-Brown.)

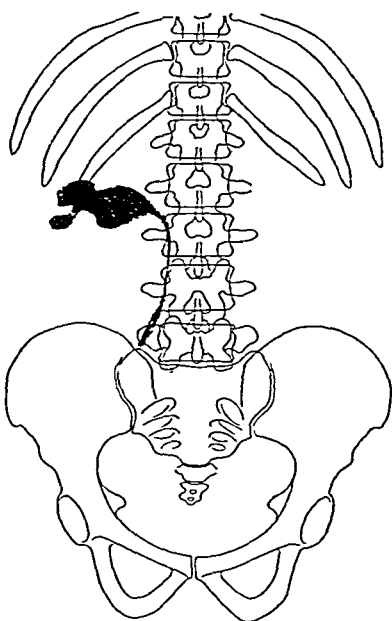


FIG. 456.—Closed cyst of the lower pole of the right kidney. The cyst involved only a centimetre or two of the kidney substance. The kidney and ureter have moulded themselves around the cyst. Cf. Fig. 454. The calices are clubbed. (After Flynn.)

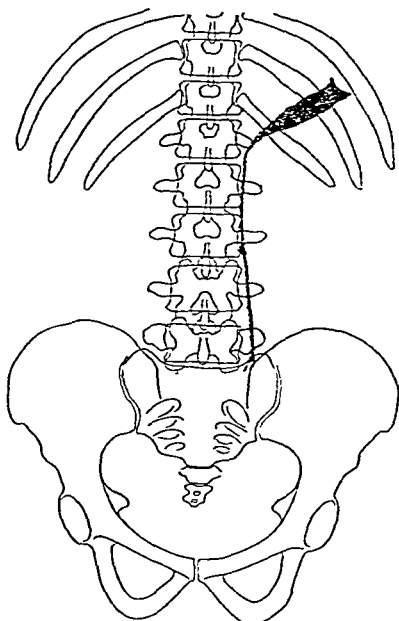


FIG. 457.—Cyst of the lower pole the size of a "child's head". The whole kidney was pressed hard up against the diaphragm and the opaque medium could not be forced into the calices. After removal it was found that the upper part of the kidney was entirely normal. The cyst was a closed one. (After Vedenski.)

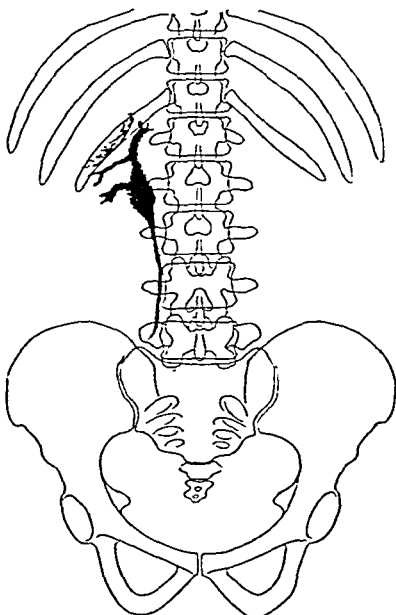
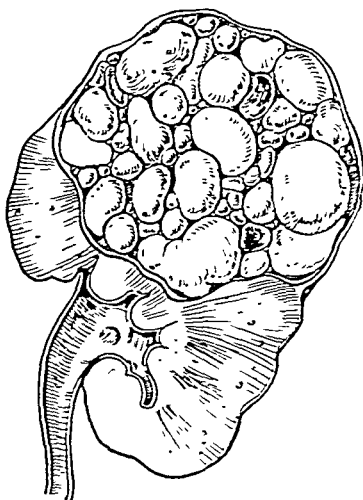


FIG. 458.—Closed cyst of the upper pole. The lower margin of the cyst is calcified. The upper and middle calices have been pushed apart and form an obtuse angle with each other. Cf. Figs. 442 and 443. The figure on the right shows the excised kidney. (After Kretschmer.)



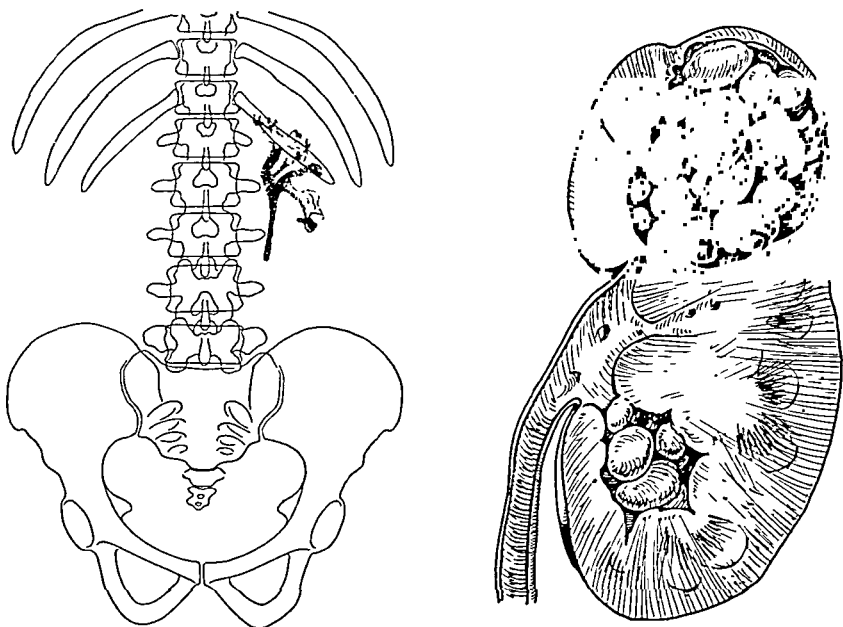


FIG. 459.—Pyelogram of left kidney which contained one primary cortical cyst which had ruptured into the middle calyx and is seen to end abruptly, while a flame-like shadow passes up from it. Insufficient fluid was used to outline the cavity of the cyst. There is also a cyst in the upper calyx, which is interrupted by it, though two normal minor calyces appear above. There is also a secondary cyst in the lower calyx which appears as a filling defect. The figure on the right shows a section of the kidney (semi-diagrammatic).

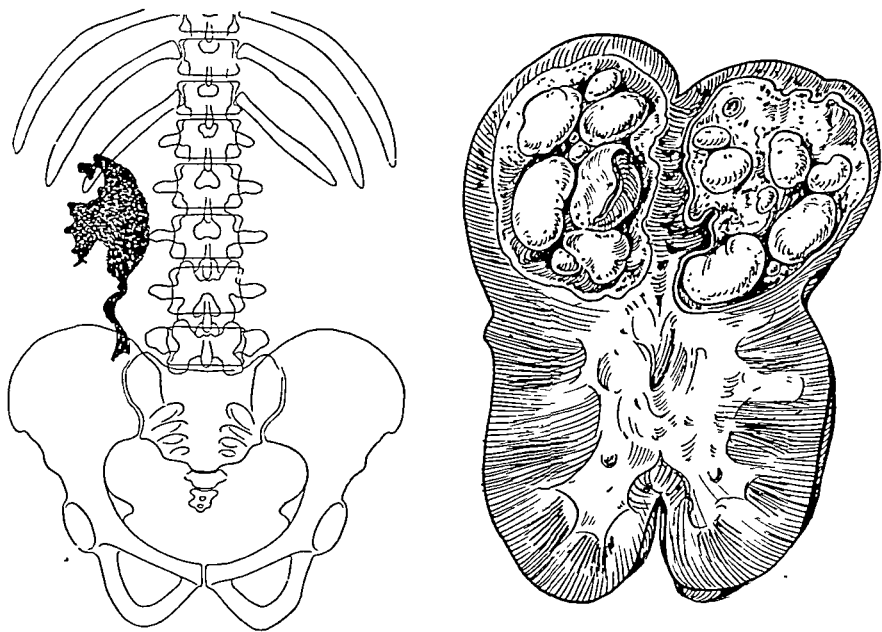


FIG. 460.—Open univesicular cyst of the upper pole of the right kidney. One of the upper minor calyces is obliterated; the other bent downwards. The pelvis is dilated. There had been for a long period repeated attacks of renal colic with the passage of hydatid sand. The figure on the right shows the excised kidney. (After Huffman.)

with each other (*Figs. 442, 458*). In these cases of completely closed cyst, however, the diagnosis cannot be made from the pyelogram alone. Pyelography offers confirmatory rather than diagnostic evidence.

Multiple Cysts.—Of the four personal cases encountered, in two only was it possible to make urological investigation. These two were examples of multiple cysts. In the first there was an open cyst of the cortex, with rupture into the middle calix, and secondary calix cysts in the upper and lower major calices (*Fig. 459*). In the second there were two primary cortical cysts each containing intact parasites; the upper cyst did not communicate with the pelvis direct but through the lower cyst, which was in communication with the middle calix (*Figs. 442, 443*).

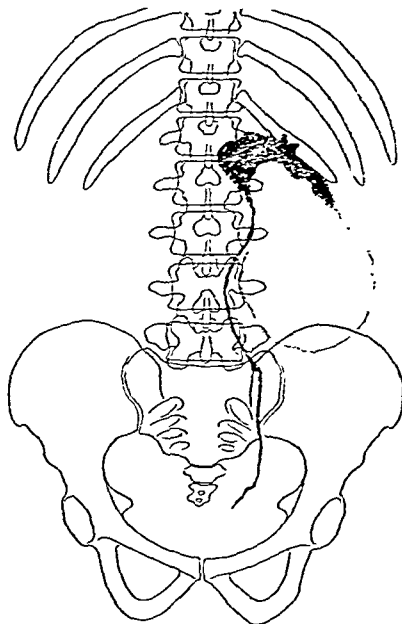


FIG. 461.—A pararenal cyst, illustrating the distortion of kidney and ureter which can occur even when the organ itself is not involved directly. (*After Surrao and Mezzera.*)

Secondary Calix Cysts.—The characteristic of a pyelogram of a calix cyst is the presence of well-defined minor calices separated from the lumen of the major calix by a filling defect representing the parasite, with or without flame-like shadows connecting the two. In such cases the urine from the intact papillæ seeps down between ectocyst and the calix wall, which is also the pericyst (*Fig. 459*). Calix cysts are always secondary, and the pyelogram will be influenced also by the presence of the primary cortical cyst.

In practice, pyelography in hydatid disease is chiefly of value in determining the side, site, and nature of the lesion in open cases already diagnosed by the identification of hydatid material in the urine, or in closed or pseudo-closed cases in which the typical ring shadow seen in the plain X-ray plate may indicate a lesion of the spleen, liver, or kidney, or a pararenal cyst. The differential diagnosis from tumour, solitary cyst, etc., is made chiefly on other grounds, especially when the hydatid cyst is so completely dissociated from the kidney pelvis that the wine-glass

sign or its modifications are absent. Yet the following points may be noted :—

1. Apart from the involved calix the pelvis is strikingly normal. In tumour this is rarely so. Seldom in cases of hydatid cyst do the calices show clubbing, though the pelvis and ureter may be dilated when cysts and membrane have been passed over a long period. Exceptions to this rule, however, may occur. (See Figs. 454, 456.)

2. If the kidney is displaced downwards by a large cyst it swings on the pelvi-ureteral junction as on a hinge, the latter remaining in its normal situation.

3. In tumour cases the ureter may be kinked, but I have not seen this occur in illustrations of hydatid cases.

Pararenal and Extrarenal Cysts.—It is important to remember that deformities in the pyelogram may occur with large cysts even when these do not arise from the kidney itself. Vedenski illustrated a case of large intraperitoneal cyst in which the middle calix of the right kidney was obliterated and the other two calices displaced. Fig. 461 illustrates a pelvic deformity from the pressure of a pararenal cyst. As a rule, however, while such cysts may displace the kidney and its pelvis *en masse*, they seldom affect the actual shadow detail. In any case they are much rarer than true renal cysts.

SUMMARY

1. The pyelographic characteristics of the various types of renal hydatid cysts are discussed and illustrated.

2. The main types are :—

a. The *pseudo-closed*, in which the cyst communicates with the renal pelvis but the parasite is intact.

b. The *open*, in which the envelope of the parasite has ruptured.

c. The *closed*, in which the cyst does not communicate with the pelvis.

d. Secondary calix cysts.

3. The effect of extrarenal cysts on the contour of the pelvis is illustrated.

A few score of cases of renal hydatids occur each year throughout the world. Few are reported and fewer still adequately investigated in a urological sense. This paper has been possible only through the careful reports and illustrations of single cases and short series of workers in many countries. Acknowledgement is made to the authors and the original contributors, in the figure captions and bibliography respectively, for the use of illustrations. The interpretation given in each case has been my own, and differs in some instances from opinions expressed by the authors. In addition, I have to express my sincere thanks to Dr. Bruce Mackenzie, of Auckland, for providing the hitherto unpublished pyelogram shown in Fig. 444.

BIBLIOGRAPHY

- BEGG, R. C., *Brit. Jour. Surg.*, 1936, xxiv, 18.
 CRAIG, G., and LEE-BROWN, R. K., *Surg., Gynecol. and Obst.*, 1928, xlii, 668.
 FLYNN, R., *Austral. and N.Z. Jour. Surg.*, 1932, ii, 99.
 HUFFMAN, L. F., *Jour. of Urol.*, 1926, xvi, 197.
 HURLEY, V., *Austral. and N.Z. Jour. Surg.*, 1933, ii, 326.
 KRETSCHMER, H. L., *Surg., Gynecol. and Obst.*, 1923, xxxvi, 196.
 LEE-BROWN, R. K., *Jour. of Urol.*, 1930, xxiii, 611.
 RAČIĆ, J., *Zeits. f. Urol.*, 1931, xxv, 341.
 ROCHE, E. H., and MACKENZIE, H. B., *N.Z. Med. Jour.*, 1935, xxxiv, 270.
 SURRACO, L. A., and MEZZERA, H., *Rev. med. latino-amer.*, 1931, xvi, 1453.
 VEDENSKI, D., *Zeits. f. Urol.*, 1928, xxii, 694.
 WOODMAN, G. S., *Brit. Jour. Urol.*, 1931, iii, 299.

MULTIPLE VILLOUS PAPILLOMATA OF THE GALL-BLADDER

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SIMPLE papillomata of the gall-bladder are extremely rare. In a recent critical review, Kerr and Lendrum¹ accept only seven examples as true simple papillomata of gall-bladder mucosa; three of these showed multiple growths, regarded as due to transplantation of fragments, analogous to what occurs in the urinary tract. Accordingly the following case appears to form a worthy addition to the latter group.

CLINICAL HISTORY

A retired farmer, aged 71, suffered from flatulent dyspepsia and shortness of breath for about six years. Early one morning he was seized with acute epigastric pain and vomiting, and gave a history of a similar attack four years previously. He was seen by one of us (F. R. B.) about twenty hours after the onset; the temperature was then 100·4° F., pulse 100, respirations 30, and there was marked cyanosis of the face and lips.

Examination revealed a large tender pyriform swelling extending from beneath the right costal margin to the right iliac fossa. A diagnosis of acute obstructive cholecystitis was made and immediate operation advised. Under gas and oxygen anæsthesia the abdomen was opened by a right rectus incision and the peritoneum found to contain a considerable amount of free fluid. A large acutely inflamed gall-bladder was removed without difficulty.

In spite of a degree of breathlessness and cyanosis, progress was at first satisfactory, and by the seventh day the temperature had fallen to 98° F., respirations 20, pulse 80. The abdominal wound healed well and the bowels moved satisfactorily. On the eighth day troublesome hiccup appeared, and there was hæmoptysis followed by increasing cyanosis and signs of consolidation in the lungs, regarded as due to infarction. During the next few days signs of increasing cardiac weakness developed, and following repeated pulmonary infarctions death occurred on the twentieth day after operation. Permission for autopsy was not obtained.

PATHOLOGICAL SPECIMEN

The gall-bladder was opened after operation and was found to contain numerous calculi and turbid purulent fluid in which there were flakes of fibrin. The fluid was not bile-stained. Further examination was delayed until after fixation. The viscus then measured 17 cm. from fundus to neck and about 5 cm. in diameter about the middle. The wall is much thickened and is œdematous.

The mucosa has almost lost its honeycomb structure and is in most places covered by a layer of fibrinous exudate, in places firmly adherent to the mucosa, in other parts only loosely attached, and separating readily to leave a red acutely inflamed surface. In the layer of fibrin, numerous small darkly-coloured calculi of the usual mixed type are seen (*Fig. 462*). At the fundus a small papillomatous tumour projects from the anterior or free wall, while from the hepatic wall just below the middle a large growth measuring 3.5 cm. in diameter projects 2 cm. into the lumen. Two smaller papillomata, one measuring 1.7 cm., the other about 0.5 cm. in

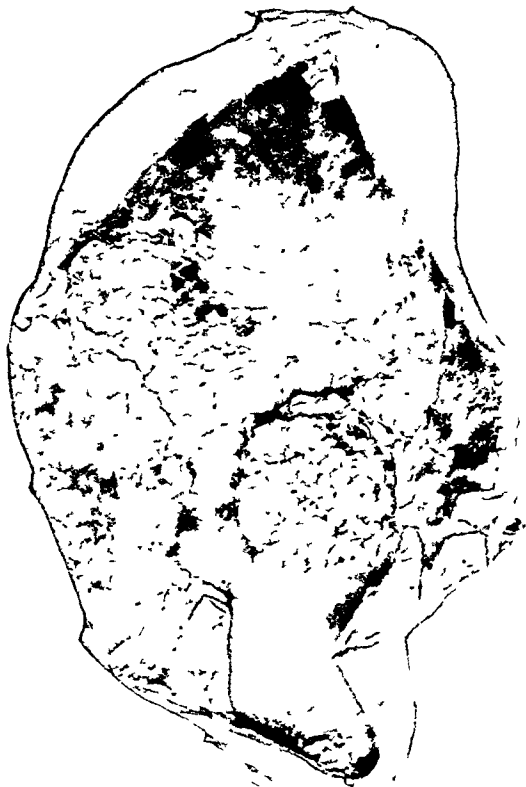


FIG. 462—Gall-bladder laid open along the free surface, showing the acutely inflamed mucosa covered with fibrinous exudate in which are embedded a few small stones. A small papilloma is seen suspended from the fundus, a larger papilloma projects from the hepatic wall of the viscus, and a smaller tumour lies to the right, close to the sectioned edge ($\times \frac{1}{2}$)

diameter, are seen on the anterior or free wall shortly below the largest growth. Each of these growths is united to the gall-bladder wall by a narrow pedicle in which there is nothing to suggest malignant infiltration. The surface of each growth presents a nodular mulberry appearance, and in places the growth can be clearly seen to consist of both blunt and fine processes closely applied together.

The calculi number about 300; they are readily graded into three sizes, 10 being approximately 1 cm. in diameter, about 50 measure 7 mm., while the remainder are fairly uniformly about 4 mm. in diameter. On section the calculi show the typical laminated structure of the so-called 'infective' stones, with pigmented nuclei and alternate layers of pigment and cholesterol.

Microscopic Examination.—Microscopic examination shows the wall of the gall-bladder to be the seat of chronic cholecystitis in an acute exacerbation. There is marked fibrous thickening and œdema of the wall, especially between the serous and muscular layers. Internal to the muscular layer the changes are more acute, and widespread hæmorrhage and purulent infiltration are apparent. The surface layer of fibrinous exudate is in places continuous with fibrinous exudation within the substance of the wall (*Fig. 463*), but elsewhere it is separated from the wall by a layer of purulent infiltration beneath which in places the epithelium persists.

Penetration of the wall by outgrowths of the lining epithelium—the so-called Aschoff-Rokitansky sinuses, often prominent in chronic cholecystitis—is scarcely



FIG. 463—Complete section of the largest papilloma, showing the small single stalk and the complex villous structure of the growth. Note the absence of invasion of the base of the stalk. ($\times 3$.)

apparent, the crypts being very shallow and extending only a short distance into the muscular coat. There is nothing to suggest that the wall in general has been the seat of proliferative epithelial changes as in cholecystitis glandularis proliferans (King and MacCallum²).

The tumours are of similar structure and each consists of a fine connective-tissue core greatly branched and expanded into numerous processes, all of which are covered by mucous membrane of simple or complex glandular type. Each papilloma is attached to the wall by a single narrow stalk consisting of a skein of dilated thin-walled blood-vessels enveloped in a fine layer of connective tissue, the surface being lined by a single layer of tall columnar cells. The complex pattern of the tumours is produced by repeated branching of the stalk, resulting in very delicate villous processes with a narrow highly vascular core covered on all sides by columnar or cubical epithelium (*Figs. 464, 465*). Between the capillaries and the epithelium lies a delicate network of reticulum fibres, closely applied to the

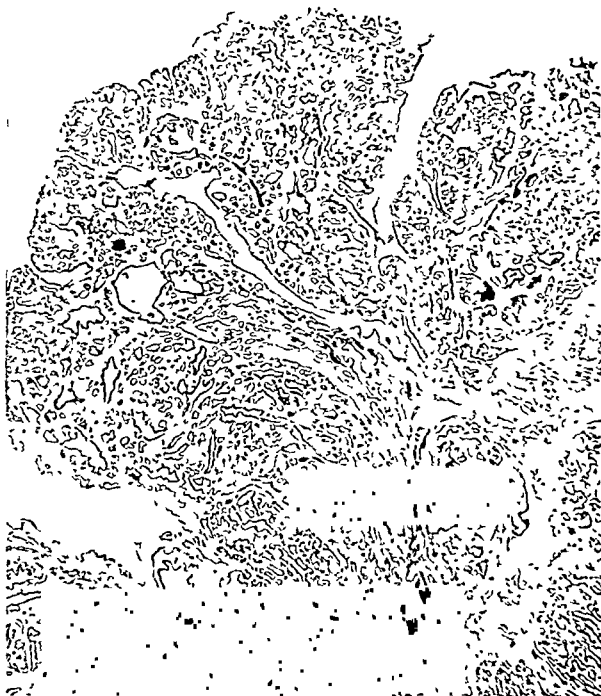


FIG. 464.—Low-power view of one of the blunt nodular processes of one of the smaller papillomata, showing the complex glandular character of the covering mucosa. ($\times 10$)



FIG. 465.—One of the early subdivisions of the stalk of the largest papilloma, showing the simple villous character with multiple branchings. ($\times 40$)



FIG. 466.—A fine villous process showing the columnar character of the epithelium and the very delicate stroma rendered prominent by silver impregnation. ($\times 85$.)

vascular endothelial cells (*Fig. 466*). The broader processes which give the surface of the tumours their nodular mulberry appearance consist of a fine vascular core covered by a more complex epithelium in which there are numerous simple test-tube-like glands, lined by columnar or cubical cells (*Fig. 464*). The structure here recalls that of simple rectal papillomata. Mucicarmine colours the free margin of the majority of the lining cells, as in normal gall-bladder epithelium, but goblet cells are entirely absent, and the surface of the epithelium and glandular spaces exhibit much mucus, together with epithelial debris and purulent material. Staining by a variety of methods, as recommended by Kerr and Lendrum, fails to reveal intracellular granules such as they describe.

Accordingly, such specialized elements as Paneth cells and entero-chromaffin cells are absent, and there is nothing in the structure of the epithelium to suggest an origin in heterotopic intestinal epithelium. In each tumour the base of the stalk is quite free from invasion by epithelial cells, and the tumours are therefore to be classified as non-malignant complex villous papillomata arising from the epithelium which normally lines the gall-bladder.

All the tumours are of similar histological structure, and it is not possible to state whether they are growths of independent origin or whether the multiplicity is due to transplantation of tumour fragments such as is commonly believed to occur in the urinary passages.

DISCUSSION

In view of the recent survey of the subject by Kerr and Lendrum, it is unnecessary to do more than report the present case. We regard the condition as one of multiple villous papillomata of the gall-bladder occurring in association with long-standing cholecystitis. We do not consider that our specimen falls into the group of cholecystitis polyposa (cholecystitis glandularis proliferans—King and MacCallum²), and with this opinion Kerr and Lendrum concur.³

SUMMARY

A case of multiple villous papillomata of the gall-bladder is described. The condition was unsuspected clinically, the gall-bladder being removed on account of acute obstructive cholecystitis.

REFERENCES

- ¹ KERR, A. B., and LENDRUM, A. C., *Brit. Jour. Surg.*, 1936, xxiii, 615.
- ² KING, E. S. J., and MACCALLUM, P., *Ibid.*, 1931, xix, 310.
- ³ KERR, A. B., and LENDRUM, A. C., *Personal communication*.

A CASE OF RUPTURED ANEURYSM OF THE SPLENIC ARTERY WITH RECURRENCE

BY CLIFFORD G. PARSONS, BIRMINGHAM

INSTANCES of ruptured aneurysm of the splenic artery are sufficiently uncommon to make their publication of interest and perhaps of some value. Between seventy and eighty cases have been reported in the literature, but in only two of these was the condition recognized pre-operatively. The following note concerns a case which appears to be unique in that successful operation on a ruptured aneurysm was followed two years later by recurrence, which, although diagnosed before operation, provided such severe technical difficulties that surgery was unavailing and the outcome fatal.

CASE REPORT

HISTORY.—The patient, a chauffeur aged 45 years, was digging in his garden when he was suddenly seized with cramp-like pain in the epigastrium. He went into the house feeling faint, but was unable to lie down because of the pain which doubled him up. His wife stated that he was pale, sweating, and complained of flatulence. He took some sodium bicarbonate and some brandy, which made him vomit. Subsequently the pain became easier and he was able to lie down. An hour later he attempted to go upstairs to bed, but felt faint and collapsed with intense pain in the left side of the abdomen. Violent pains occurred about every two hours, radiating up to the left shoulder and down the arm. During the night the pain disappeared, and for the following two days he remained in bed almost free from pain. On the third day he got up and on the fifth returned to work. That afternoon, whilst sitting down, he was again seized with pain in the left hypochondrium radiating to the left shoulder and arm, the pain being acute and stabbing in character, lasting for about ten minutes, and leaving behind it a dull ache. On this occasion he did not feel faint. Throughout the night attacks of pain occurred every two hours. An enema was given on the following morning, but since no relief was obtained he was admitted to the General Hospital, Birmingham, on Oct. 18, 1934.

ON ADMISSION.—When first seen his temperature was 98.5° and his pulse 85 per minute. He was in a condition of shock, his abdomen was distended, and intensely rigid and tender, especially in the left lumbar region. The abdomen was hyper-resonant to percussion, and the reflexes were present. A diagnosis of perforated gastric ulcer was made.

FIRST OPERATION.—Immediate operation was performed by Mr. B. T. Rose, the abdomen being opened through a right paramedian incision. The peritoneum was full of bright-red blood; the abdominal viscera were all normal, but there was much blood-clot in the splenic area, from which the bleeding appeared to be coming. A second incision was made along the left costal margin, and disclosed arterial bleeding from an aneurysm situated in the middle third of the splenic artery. Ligature of the artery was carried out proximal to the aneurysm, and the spleen and its pedicle removed. A gum saline infusion was given.

Convalescence was uneventful except for mild jaundice, which developed on the day after operation. Professor Haswell Wilson reported as follows on the pathology of the removed spleen. "I can see no evidence of syphilis. The spleen itself is rather fibrotic and there is extensive hyaline degeneration in the smaller vessels. The splenic artery is fibrous and hyaline and there is some increase in the elastic tissue." The Wassermann reaction was negative.

SUBSEQUENT PROGRESS.—The patient remained quite well until the beginning of January, 1936, when he complained of flatulence and a dull aching pain in the left hypochondrium.

He was seen by Mr. B. T. Rose a fortnight later, and was found to be tender below the left costal margin. He was constipated and had vomited on the previous day, the pain thereby being relieved. A diagnosis of gastric ulcer was entertained, and an X-ray of the stomach showed an "ulcer at middle of lesser curve". He was treated on medical lines for this complaint by Dr. J. M. Smellie and remained well until Aug. 22, when he again complained of



FIG. 467.—Aorta and aneurysm laid open from the front, showing dilated celiac axis. The rest of the specimen consists of false aneurysmal sac embedded in pancreas.

pain in the left side of the upper abdomen. The pain was not very severe, and was intermittent, having no apparent relationship to food, and being unrelieved by diet or alkalis. During the following three or four days the pain became worse, and on Aug. 30 he vomited bile-stained fluid on four occasions. The pain was more severe on the following day, but was relieved by lying down. While getting up on Sept. 1 he felt faint and again vomited bile-stained fluid. He complained of left-sided pain, and called in his doctor, who ordered his transfer to hospital.

ON RE-ADMISSION.—He was admitted on the same day under Professor L. G. Parsons, and was found to be pale, cold, collapsed, and sweating; temperature 96.2° , pulse 100, of small volume. He was in no way apprehensive, lying comfortably in bed untroubled by pain. Soon after admission he vomited some brownish fluid, which gave a strongly positive reaction to the benzidine test. His abdomen was distended in its lower part, was not rigid, but was tender in the left hypochondrium. The left loin was somewhat dull, and the rest of the abdomen tympanitic to percussion. He was constipated, and the urine contained a cloud of albumin and numerous pus cells. Any movement in bed gave him intense abdominal pain. Three hours later his pulse had risen to 138, and a blood transfusion was given. His pulse immediately fell to 80, but he complained that he had pain in the left hypochondrium, and a few minutes later had severe pain in the left shoulder radiating down the left arm. The tenderness in the left side of his abdomen increased, and he soon complained of tenderness in the right iliac fossa. There was by now definite dullness in the left flank which 'shifted' when the patient was rolled on to his side. In the next hour or so the pulse rose to 120, and Mr. B. T. Rose saw the patient and decided to operate, remarking that the physical signs reminded him of those in a woman bleeding from a ruptured ectopic gestation.

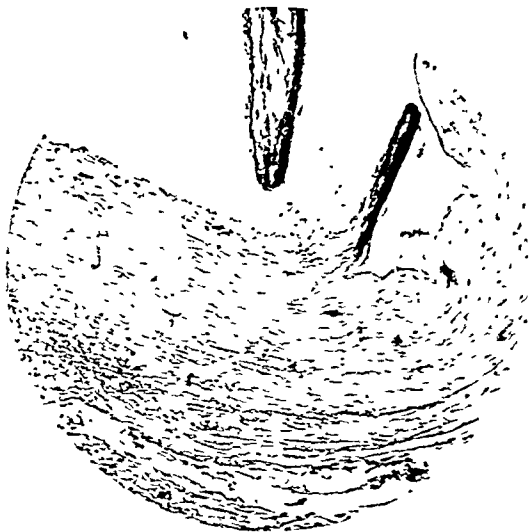


FIG. 468.—Microphotograph of portion of wall of splenic artery showing fibrosis, hyaline degeneration, and round-celled infiltration. Calcified plaque in intima.

SECOND OPERATION.—The peritoneum was again full of blood; furious arterial bleeding was taking place from an aneurysm of the splenic artery. This aneurysm, which was the size of a cricket ball, lay in the pancreas and had eroded into the lesser sac of the peritoneum. As it was quite impossible to stop the bleeding by obliterating the aneurysmal sac, which appeared to lead directly off the aorta, the cavity was tightly packed and the wound closed. The patient died on the following morning.

AUTOPSY.—At autopsy the peritoneum was found to be full of blood-clot, the older portions of the clot being in the lesser sac and the right paracolic gutter. The greater part of the body of the pancreas was occupied by a gaping false aneurysmal sac, which, bulging upwards and forwards, was adherent to the mid-portion of the lesser curve of the stomach. The floor of the aneurysmal sac consisted of fibrotic pancreatic tissue and was partially lined by laminated ante-mortem clot. An atrophic splenic artery could be traced in the wall of the false aneurysmal sac (i.e., beyond the point at which the artery had been ligated in the first operation). The splenic artery between the coeliac axis and the site of ligation was dilated and had atrophic walls containing calcareous atheromatous plaques. The coeliac axis was the seat of a small aneurysmal dilatation; the wall was thin and contained calcareous material. The other organs and arteries were essentially normal, and no sign of gastric ulcer could be demonstrated. (Fig. 467.)

Professor Haswell Wilson and Dr. S. Macdonald reported as follows on the microscopic findings. "Nothing suggestive of syphilis or periarteritis nodosa is seen in any of the sections. There is extensive fibrosis of the media (of the splenic artery), with much hyaline degeneration and occasional small areas of degeneration. The intima is thick and fibrous, and there is a number of atheromatous plaques with calcification (*Fig. 468*). Beneath one of these the media is much thinned, and it is in relation to this that the aneurysmal dilatation has occurred. In the peri-arterial tissues there is a considerable amount of round-celled infiltration. This encroaches on the adventitia and also surrounds some islands of atrophied pancreatic tissue which are embedded in the fibrous wall of the aneurysm."

DISCUSSION

The history is interesting since the symptoms closely simulate those of gastric ulcer and perforation. The pain, however, was aggravated by exercise rather than by food, and was situated rather farther to the left and lower down than the pain of gastric ulcer. Some degree of remission in symptoms was evident, but melæna did not occur, although blood was demonstrated in the vomit on one occasion. The cause of the pain is debatable. In the first place, in view of the X-ray findings it is possible that a gastric ulcer may have been present, accounting for the pain and sickness in the early part of 1936, although it could not have been responsible for the pain immediately preceding the final catastrophe, as no sign of ulcer was found at autopsy. The pain of gastric ulcer is probably due to severe spasm of the muscular wall of the stomach, and since the aneurysm was adherent to the lesser curve it is possible that by irritation it produced a similar type of spasm. On the other hand, the most likely explanation of the pain and intermittent character of the symptoms is that the aneurysm leaked at certain times into the lesser sac and set up a mild peritonitis, which in turn led to the formation of adhesions, and the building up of the false aneurysm. The march of symptoms in the acute attacks—pain in the left hypochondrium, then in the left shoulder, and finally in the right iliac fossa—strongly suggested a peritonitis of the lesser sac with overflow through the foramen of Winslow into the right paracolic gutter, and enabled a correct diagnosis to be made before the second operation. Despite the erosion of the pancreas there was no pain in the back.

This case is reported at Mr. Rose's suggestion, not so much for its clinical and diagnostic importance as on account of its rarity. The diagnosis is obviously fraught with difficulty, since in many cases the disease may be symptomless until rupture takes place. Preliminary symptoms when present are generally suggestive of gastric ulcer, for there is epigastric or left hypochondriac pain, which may on occasion be post-prandial; vomiting is by no means uncommon and may be severe; and both hæmatemesis and melæna have been described. In some instances a tumour had been felt, and in one case, as fatty indigestion was present, a diagnosis of chronic pancreatitis was made.¹ An excellent review of the literature and symptomatology is given by Anderson and Gray.² Should the condition be suspected various confirmatory diagnostic procedures have been recommended: radiography has been employed with success³; pancreatic deficiency may be present⁴; a bruit audible with the stethoscope in the left hypochondrium has enabled the condition to be diagnosed before operation, and demonstrates the value of abdominal auscultation.^{3, 4, 5}

Rupture commonly occurs in two phases, and the importance of making the diagnosis and operating during the first phase is emphasized by Brockman, who writes as follows: "A primary rupture takes place into the lesser sac or splenic

pedicle. This is not usually fatal since clotting occurs in a short time . . . Later, after an interval of two or three weeks, the secondary rupture takes place with a rapidly fatal termination". Erosion into the lesser sac is much commoner than rupture into the gut. The symptoms are those of the 'acute abdomen', and may simulate those of perforated gastric ulcer, acute intestinal obstruction, acute pancreatitis, or pulmonary embolism. Pain of intense severity and of sudden onset is felt in the left side of the abdomen or the pit of the stomach. It may double the patient up, and is frequently accompanied by persistent vomiting. A severe degree of shock, a rapid pulse, pallor, with a varying degree of rigidity, and some degree of dullness in the flanks, particularly the left, are the most usual findings, and combine to give a picture of internal hæmorrhage remarkably like that of a ruptured ectopic gestation. If the abdomen is opened by the usual right-sided laparotomy incision the source of bleeding may easily be missed, and although the hæmorrhage may cease temporarily, secondary and fatal rupture will take place a few days later.

The causation of aneurysms of the splenic artery is somewhat obscure, and atheroma, syphilis, chronic infection, embolism, and trauma have all been blamed as etiological factors. Of these atheroma is probably the condition most frequently found, as in the present instance, in which the disease was remarkably localized.

REFERENCES

- ¹ LOWER, W. E., and FARRELL, J. I., *Arch. of Surg.*, 1931, xxiii, 182.
- ² ANDERSON, W., and GRAY, J., *Brit. Jour. Surg.*, 1929-30, xvii, 267.
- ³ HOGLER, F., *Wien. Arch. f. inn. Med.*, 1920, i, 543.
- ⁴ BROCKMAN, R. ST.L., *Brit. Jour. Surg.*, 1929-30, xvii, 692.
- ⁵ GOODHEART, T. F., *Trans. Pathol. Soc. Lond.*, 1889, xl, 67.

ONE-STAGE LOBECTOMY FOR HYDATID DISEASE
OF THE LUNG*

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CASE REPORT

HISTORY.—Mrs. C. E., aged 60, was referred to the Surgical Unit of the Welsh National School of Medicine by Dr. Melville Hiley, of the Welsh National Memorial Association. She had suffered from a recent exacerbation of a chronic cough, and for three months previous to admission had expectorated blood-stained sputum; since the age of 18 she had had a slight cough. During the three months' illness she had lost 2 stone in weight. There was no pain in the chest; dyspnoea on exertion was present; and there had been some œdema of the feet and ankles which followed attacks of palpitations.



FIG. 469.—Lipiodol bronchogram. Note displacement of bronchial tree by a circular swelling in the left lower lobe. The arrows indicate the periphery of the rounded shadow, the details of which are obscured by the associated pleural effusion.

ON EXAMINATION.—She was a thin wiry woman. The temperature and pulse-rate were normal. There was marked dullness to percussion over the base of the left lung, with diminished movement of the overlying ribs; the physical signs were those of a pleural effusion.

X-ray Examination.—The lower lobe of the left lung appeared to be opaque, and there was fluid present with a typical outer concave edge reaching upwards towards the axilla.

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A lipiodol bronchogram gave a peculiar picture, the terminal branches of the lower bronchi being flattened and distorted by a circular shadow in the lower lobe. The right bronchial tree appeared normal (*Fig. 469*).

The diagnosis of lung cyst, probably of hydatid origin, was inferred, but the recent history and the presence of pleural effusion suggested the possibility of a new growth. The blood-count was normal, there being no eosinophilia: the Casoni reaction was not done. Exploratory thoracotomy was advised and accepted by the patient.

OPERATION (Sept. 29, 1936).—Under general intratracheal gas and oxygen the chest was opened in the sixth intercostal space. On incising the pleura, free yellowish fluid escaped; a firm spherical mass was felt in the left lower lobe which was free from adhesions except to the diaphragm, where stout fibrous bands required division and ligature. The lobe was removed by the one-stage lobectomy technique employing a Roberts-Nelson tourniquet for

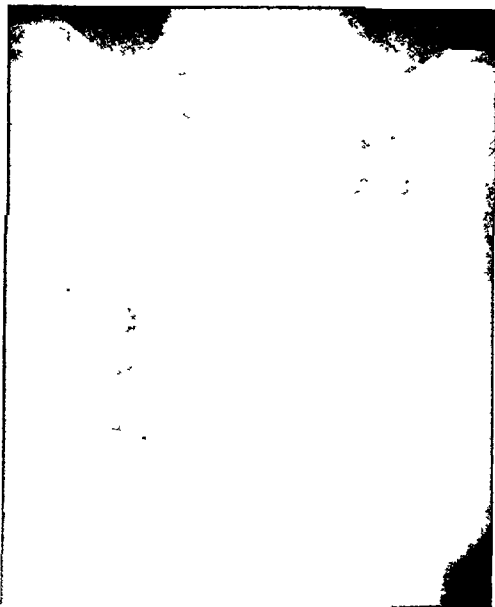


FIG. 470—Radiograph of chest taken by a portable X-ray apparatus three days after the operation. The lung has expanded, but there is fluid present at the left base. This fluid rapidly became absorbed and did not require aspiration.

the pedicle, which was then divided and secured by interrupted catgut sutures. Air-tight drainage was provided by a tube leading through a stab wound in the seventh intercostal space to a water-seal drainage system. The chest was closed completely with four pericostal sutures and two layers of continuous catgut and silkworm sutures in the skin.

SUBSEQUENT PROGRESS.—This was satisfactory; about 12 oz. of blood-stained fluid escaped into the bottle for the first forty-eight hours and then ceased. The tube was then removed, the patient being radiographed in bed every other day to watch the rate of lung expansion and to guard against the unsuspected collection of fluid. A little fluid formed, but was quickly absorbed (*Fig. 470*). The wound healed by first intention (*Fig. 471*). The patient was allowed up on the eighteenth day, and discharged home on the twenty-sixth day after operation, well and symptom free. The equanimity of a splendid patient contributed largely to this smooth and happy convalescence.

PATHOLOGICAL REPORT.—The lobe was hardened and then bisected; a typical large hydatid cyst was present, compressing the surrounding lung tissue, part of which formed a well-marked adventitious capsule (*Fig. 472*). No daughter cysts were present. Typical scolices were present in the scrapings from the inner wall of the cyst.



FIG. 471.—Photograph showing the main incision and the site of the stab wound twenty days after operation.

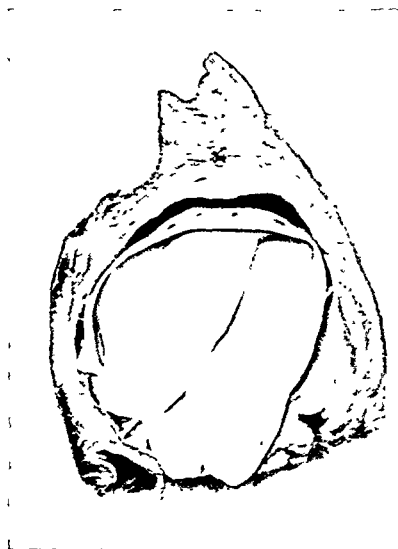


FIG. 472.—The excised lobe containing the hydatid cyst after hardening and bisection.

DISCUSSION

The correct diagnosis was uncertain until thoracotomy, despite the presence of a well-defined circular shadow on the radiograph ; the loss of weight, the hæmoptysis, and the pleural effusion, in a woman of 60, suggested carcinoma. The peripheral location of the tumour, making lobectomy feasible, induced us to advise thoracotomy.

The potential danger of hydatid cysts has been emphasized by Dew,¹ and he points out that "it is the onset of complications which alone determines the first clinical manifestation in nearly three-quarters of the cases". In my case it was the presence of pleural effusion and the threatened rupture of the cyst into the bronchus (causing hæmoptysis) that aggravated the long-existing symptom of cough. Dew believes that most hydatid cysts are as old as the host, and the persistent cough of many year's duration suggests the longevity of this particular one. The dangers of rupture are too well known to require emphasis. Such a disaster usually causes immediate generalized hydatid anaphylaxis and the development later of secondary cyst formation from implantation of the scolices. Rupture into the pleura also causes hydatid pneumothorax, which, proceeding to intra-pleural suppuration, is a complication of much gravity. Deep parabronchial cysts may, and do, rupture into bronchi, and are often followed by spontaneous cure, though there is a danger of asphyxia or subsequent suppuration. In this case the subpleural site of the cyst and the associated effusion suggested that rupture into the pleural cavity was imminent ; at several places in the specimen the laminated coat of the cyst, having perforated lung tissue, can be seen beneath the visceral pleura.

Surgical intervention in pulmonary hydatid cysts usually comprises a two-stage operation in which adhesions between the pleura overlying the cyst and that of the parietes are artificially created by the introduction of an iodine pack after the appropriate ribs have been resected ; at a later date the cyst is opened after formalin has been injected with the object of killing the scolices to prevent secondary echinococcosis developing in the surrounding tissues ; subsequently the cyst wall may be evulsed. I believe this is the first case reported in which primary lobectomy has been employed, and in suitable cases the operation should be safe and free from the peculiar difficulties and dangers of two-stage drainage operations, where the risks of spreading infection into the pleural cavity and the dangers of anaphylaxis must always exist. It is a fortunate fact that the majority of hydatid cysts of the lung are situated in the lower lobes and are sub-pleural.

The comparatively slight disturbance to a patient of 60 caused by one-stage lobectomy is noteworthy ; the easy convalescence was due no doubt to the rapid healing of a non-infected pedicle. By contrast, lobectomy for bronchiectasis in elderly subjects, where the mortality is immensely higher than in the young, is of greater risk, because of the presence of gross infection in the pedicle stump and the subsequent development of an empyema in a patient poorly equipped to combat it.

I am deeply indebted to Professor Lambert Rogers for his advice and encouragement ; to Mr. A. F. Goode, with whom I operated on this patient ; and to Dr. Melville Hiley, who referred the case to the Surgical Unit after preliminary investigation.

REFERENCE

¹ DEW, H., *Brit. Jour. Surg.*, xviii, 275.

THE EFFECT OF BRACHIAL PLEXUS BLOCK ON PATIENTS SUFFERING FROM SECONDARY TRAUMATIC SHOCK

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A PATIENT who has received an injury reacts to that injury according to a well-recognized clinical sequence. The immediate reaction is termed 'primary shock.' The reaction manifested after an interval, which varies from a few minutes to many hours, is termed 'secondary shock'.

Secondary traumatic shock is characterized by pallor, coldness, subnormal temperature, and rapid pulse. Whilst the blood-pressure is usually lowered, it may be raised (Fraser and Cowell¹). A constant symptom is that the patient feels ill. These features are exhibited to a varying degree in any particular case, depending upon the severity of the trauma and the physiological and psychological make-up of the patient. With the exception of the subjective symptoms one or more of these features may be absent.

The work of O'Shaughnessy and his associates² upon experimental animals affords strong evidence that secondary traumatic shock is a nervous phenomenon. The results of their cross-circulation experiments tend both to discount the importance of the liberation of a histamine-like substance at the site of the trauma as an agent in producing shock, and to minimize the effect of fluid loss.

If secondary traumatic shock is initiated and maintained by a continuous discharge of nerve impulses from the traumatized area, as is attested by these workers, it was thought that the condition of shock consequent upon a severe injury to the upper limb might be relieved by a brachial plexus block, and that this form of anaesthesia might be suitable for dealing with the trauma in such cases.

A number of brachial plexus blocks was therefore performed with the purpose of determining their effect upon the blood-pressure in a series of cases with lesions of the upper limb. Patients suffering from varying degrees of shock due to lesions of the upper limb were selected. Blood-pressure readings were taken during the course of the induction of anaesthesia and the subsequent operation.

In the following series the clinical condition of the patients served as a guide to the degree of shock. In shocked patients, whether of the common hypotensive type or of the less common hypertensive type, any agent which produces a sudden fall in blood-pressure serves to aggravate the shock, and the patient's general condition deteriorates concomitantly with this fall in blood-pressure.

Figs. 473 and 474 illustrate the effect of blocking the brachial plexus in two cases suffering from moderate shock.

CASE REPORTS

Case 1.—W. D., a well-developed boy aged 18, sustained a severe laceration and crush injury of the left forearm in a lift accident. Bleeding was severe, and a tourniquet was applied to the upper arm twenty minutes after the injury. He was admitted to hospital half an hour

after the injury. He then showed symptoms of shock. He was pale, sweating, and felt very ill. His pulse was of fair volume and its rate 80, and his temperature 98.4° F. From his statement it appeared that he had lost about a pint of blood. The wound was still bleeding; a second tourniquet was applied, and the bleeding ceased. He was treated for about an hour with hot blankets and an electric cradle, and his general condition improved.

He was then taken to the operating theatre (zero time, *Fig. 473*) and a brachial plexus block was performed. Blood-pressure readings were taken before the anæsthetic was given and were repeated at intervals as shown in *Fig. 473*. At 23 minutes 70 c.c. of 1 per cent procaine with 6 min. of adrenaline were injected into the brachial plexus. At 27 minutes, anæsthesia being complete, the second tourniquet to be applied was removed. Bleeding recommenced, and at 31 minutes the first tourniquet was removed and the wound examined. Torn and bleeding superficial veins were ligatured; deep vessels and nerves were not damaged. The wound was trimmed and loosely sutured. Throughout the operation the patient felt no pain in his arm. *Fig. 473* shows that before the anæsthetic was injected the systolic blood-pressure remained over 165 mm. Hg., the diastolic being at 85 mm. Hg. and

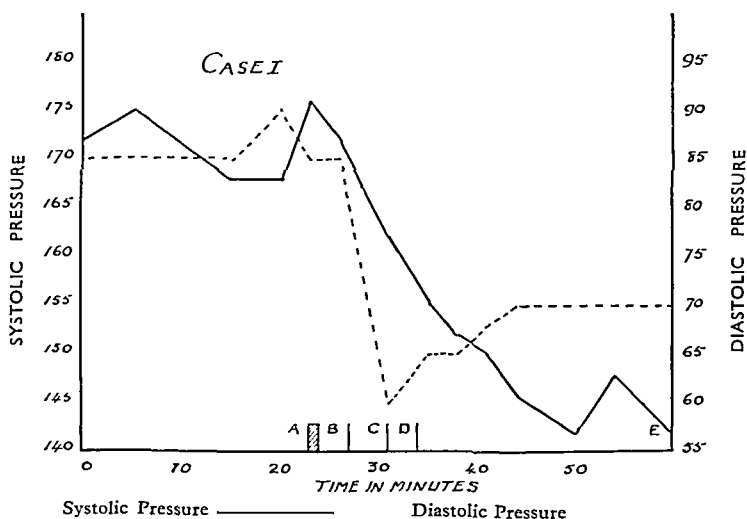


FIG. 473.—Case 1. W. D., male, aged 18. Two hours previously he had caught his arm between a moving lift and landing; severe laceration of extensor muscles of forearm; about a pint of blood lost; maceration of muscle. Clinical shock. A tourniquet had been applied in the ambulance and another one on arrival at the hospital. Plexus block was obtained by injecting into the brachial plexus 70 c.c. 1 per cent procaine and 6 min. adrenaline. A, Plexus block; B, 1st tourniquet removed; C, 2nd tourniquet removed; D, Operation begun; E, Patient returned to ward.

above. Two minutes after completion of the injection the pressure was maintained. After removal of the tourniquets, and during the operation, the patient complained of feeling faint, began to sweat, and became restless.

His systolic blood-pressure fell progressively from its previous high level to reach 142 mm. Hg. at 50 minutes. The diastolic pressure fell first to 60 mm. Hg. and then rose to 70 mm. Hg., at which level it remained. At 60 minutes the patient, still being shocked, was returned to the ward and there gradually improved. He made an uninterrupted recovery. During convalescence ten days later his blood-pressure was found to be 110/70.

Case 2.—B. H., a spare man aged 23, caught his left hand in a hydraulic press. He was admitted to hospital an hour and a half later. On admission his temperature was 98° and his pulse 84 and of poor volume. He was pale, cold, and listless, and felt very ill. His hand was X-rayed on the way to the ward, where he was treated with hot blankets and a cradle for two and a half hours.

Whilst in the ward undergoing this treatment his condition improved. An examination of the radiograph showed fractures of the 2nd, 3rd, and 4th metacarpals and of the greater multangular bone. Five hours after the injury (zero time, *Fig. 474*) he was taken to the

theatre, and, as in the previous case, blood-pressure readings were taken at intervals. *Fig. 474* illustrates the findings in this case. At 17 minutes a brachial plexus block was performed, 60 c.c. of procaine with 5 min. of adrenaline being injected into the left brachial plexus. At 26 minutes, the block proving effective against all stimuli, the wound was exposed, and it was found that there was a severe laceration down the ulnar border of the hand exposing the 5th metacarpal bone, and that the muscles of the thenar eminence had been squeezed out and lay in a pulpy mass between the index finger and the thumb. The space between the 1st and 2nd metacarpals, usually occupied by a fleshy mass, was quite empty, and the skin on the dorsal and volar surfaces of the cleft could be made to touch. Most of the extruded muscle was cut away, the lacerations trimmed, and the wounds loosely sutured. At 30 minutes, however, the patient complained of feeling unwell and called for a drink of water. His condition steadily worsened, and at 45 minutes, by which time the systolic blood-pressure had fallen from 135 mm. to 90 mm. Hg, he was semi-conscious, pale, and sweating. At 49 minutes he was given 1 c.c. of ephedrine subcutaneously, and at 53 minutes his systolic

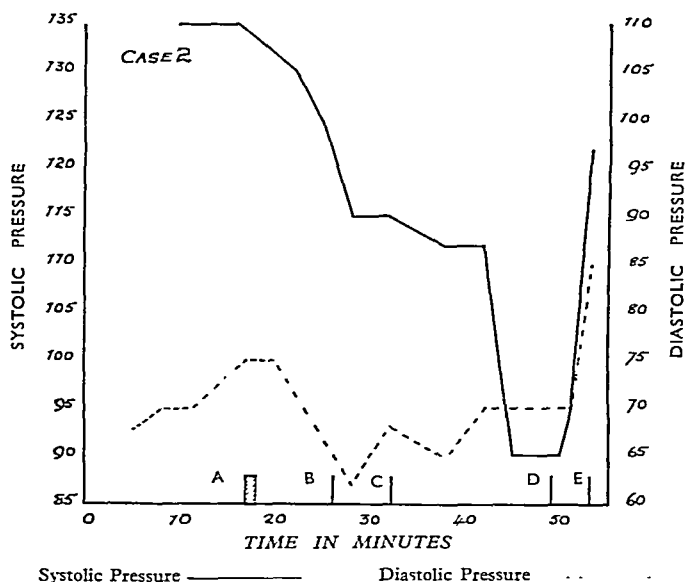


FIG. 474.—Case 2. B. H., male, aged 23. Five hours previously he suffered a crushing injury of the forearm and hand. The muscles of the thenar eminence had burst through the skin and lay between the forefinger and thumb. There was a laceration down the ulnar border of the hand through the whole skin for 3 inches. There were fractures of the 2nd, 3rd, and 4th metacarpals and of the greater multi-angular bone. Clinically the patient was in a condition of shock. Plexus block was obtained by the injection of 60 c.c. 1 per cent procaine and 5 min. adrenaline into the brachial plexus. A, Plexus block; B, Operation commenced; C, Clinical condition poor; D, 1 c.c. ephedrine injected; E, Patient returned to ward.

pressure had returned to 122 mm. Hg. At the same time his general condition improved and he went back to the ward feeling fairly well.

Despite the exhibition of anti-gas gangrene serum, infection from gas-forming organisms complicated his recovery, and it was not until three weeks later that he was discharged, quite fit and the wounds nearly healed.

DISCUSSION

In Case 1 the brachial plexus block was followed by a severe fall in blood-pressure and marked deterioration of the patient's condition. This may have been due to removal of the tourniquets, and advocates of the 'humoral theory' would see here the flooding of the circulation with a histamine-like substance over which

the brachial plexus block would have no control. It will be noticed that the fall of blood-pressure occurred in this case before the operation—débridement and suture—was begun, so that stimulation or irritation of nerve-endings at this time was not the responsible factor.

In *Case 2*, following the brachial plexus block and during the operation of débridement, the blood-pressure fell so low and the patient's condition became so critical that an injection of ephedrine was required.

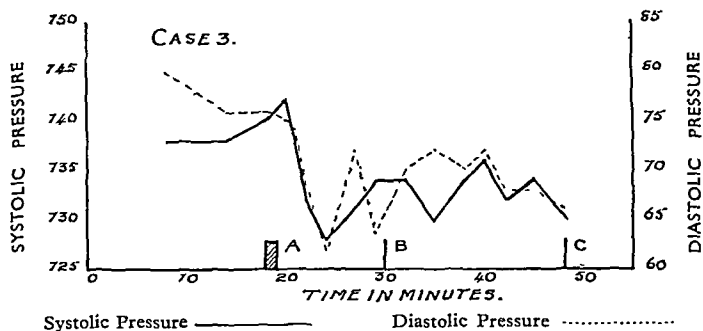


FIG. 475.—*Case 3.* R. F., male, aged 25. Three hours previously he caught his hand in a hydraulic press; the skin was scraped off the dorsum of the middle, ring, and little fingers. No clinical shock. Plexus block was obtained by injection into the brachial plexus of 60 c.c. 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Tanning started; C, Patient returned to ward.

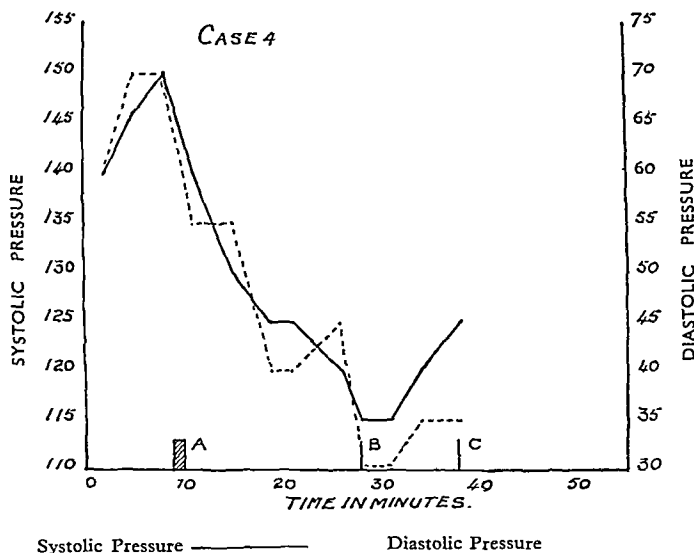


FIG. 476.—*Case 4.* J. W., male, aged 16. Friction burn of second degree on volar aspect of forearm, 15×4 in. in area. Admitted to operation theatre $2\frac{1}{2}$ hours later. No clinical shock. Plexus block by 60 c.c. 1 per cent procaine and 5 min. adrenaline injected into brachial plexus. A, Plexus block; B, Tanning begun; C, Patient returned to ward.

The unsatisfactory condition of these two patients under this form of anaesthesia, and particularly the state of *Case 2*, which before the exhibition of ephedrine gave rise to serious concern, made further observations on the effect of brachial plexus block upon shocked patients impracticable. It was considered, however, that the primary purpose of the inquiry had been served, and that the conclusion to be drawn was that brachial plexus block, far from exerting a protective influence,

seemed to aggravate the condition of shock; that although this could be controlled by ephedrine, plexus block showed no advantage over other forms of anaesthesia, and was not indicated in cases of trauma to the upper limb accompanied by shock.

It remained to investigate the cause of this fall in blood-pressure, and for this purpose readings were taken on a number of patients suffering from burns and minor injuries in whom there was no clinical evidence of shock, and who were anaesthetized by plexus block. *Figs. 475-478* illustrate the blood-pressure readings

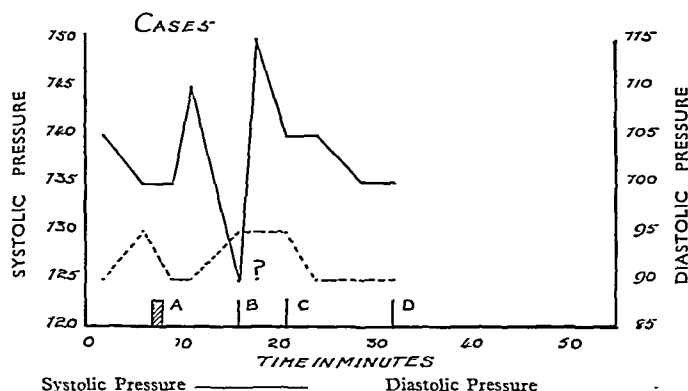


FIG. 477.—Case 5. W. W., male, aged 32. Electric burns of second degree over the dorsum of one hand received two hours previously. No clinical shock. Plexus block obtained by injection into brachial plexus of 60 c.c. of 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Cleaning; C, Tanning begun; D, Patient returned to ward.

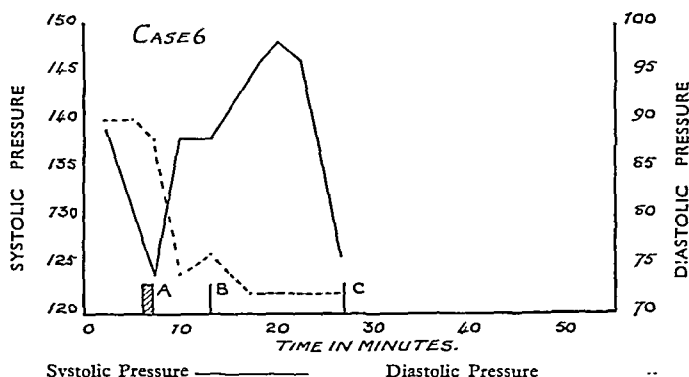


FIG. 478.—Case 6. E. H., female, aged 21. Second degree burn over one-third of dorsum of hand two and a half hours previously. No clinical shock. Plexus block obtained by injection of brachial plexus with 60 c.c. of 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Tanning begun; C, Patient returned to ward.

under these circumstances. It will be seen that in these cases the results are variable. *Case 3* shows a definite but slight fall in blood-pressure; *Case 4* a considerable and, in this instance, quite alarming fall; *Case 5* an indefinite variation; and *Case 6* a rise in the systolic pressure.

In order to eliminate the effect of shock altogether, blood-pressure readings were taken on a case in which plexus block was undertaken to determine whether a brachial neuralgia was cortical in origin (*Fig. 479*). It will be seen that a severe fall in blood-pressure followed the block and that the pressure returned to its previous value in twenty minutes.

Various explanations might be assigned to these effects:—

1. The local anæsthetic might exert a depressor effect in virtue of its pharmacological action.

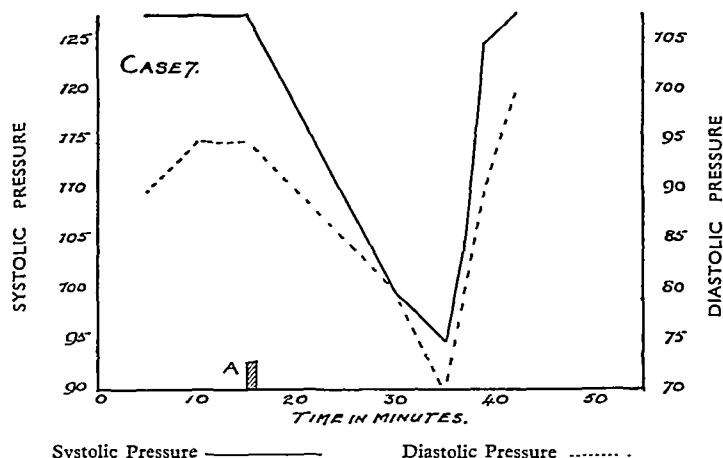


FIG. 479.—Case 7. G. T., male, aged 45, a sufferer from severe brachial neuralgia. Test block, obtained by 60 c.c. 1 per cent procaine and 5 min. adrenaline injected into brachial plexus, to see whether pain was central or peripheral, with a view to surgical treatment. Block relieved the pain. A, Plexus block.

2. If the 'nerve theory' were true, the impingement of the needle on the brachial plexus, which even if preceded by local anæsthetic causes a sharp pain down the arm, might be responsible for stimulating the plexus and producing shock.

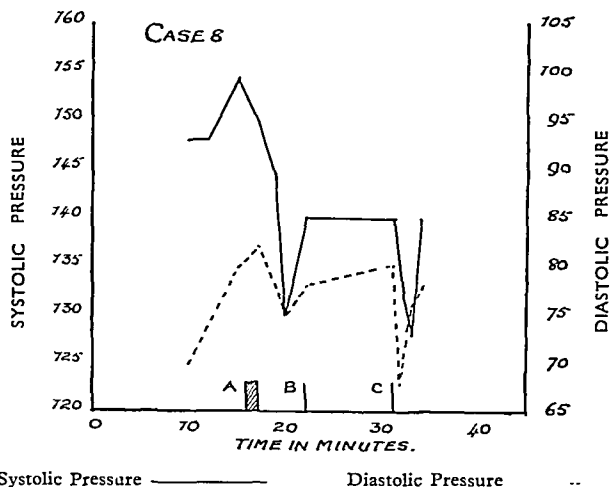


FIG. 480.—Case 8. J. B., male, aged 39. Blood-pressure readings taken during the induction of artificial pneumothorax. For local infiltration 10 c.c. of 1 per cent novocain and 2 min. adrenaline were used. A, Local infiltration; B, Pneumothorax needle inserted; C, Pneumothorax induced.

3. The needle might in certain cases pierce the pleura, producing an effect such as is shown in Fig. 480, where the blood-pressure readings are recorded during the induction of an artificial pneumothorax.

In order to determine the effect of local injection apart from brachial plexus block, blood-pressure readings were taken during the induction of anæsthesia by local infiltration into the tissues. In only two of these cases (*Cases 9 and 10*) was there any clinical shock, and here the shock was of a moderate degree of severity only.

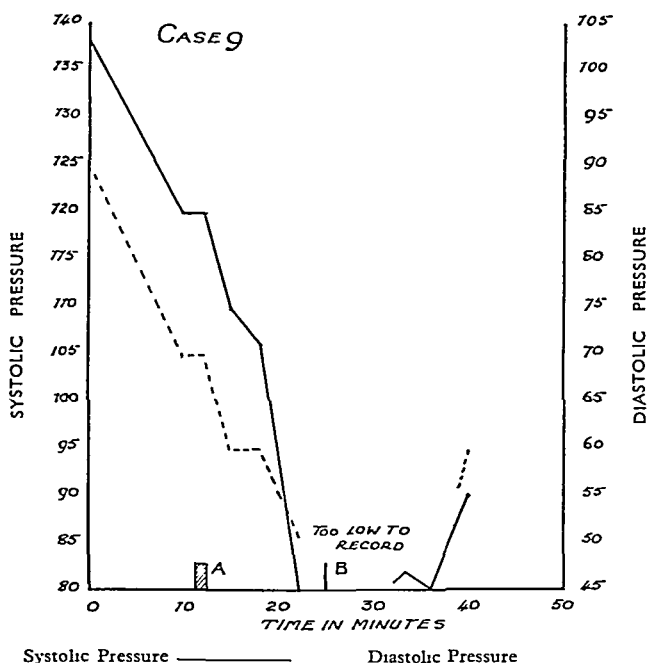


FIG. 481.—*Case 9*. B. C., male, aged 51. Severe laceration of forearm, with some clinical shock, treated by suture. Injection of 70 c.c. 1 per cent procaine with 5 min. adrenaline was made subcutaneously around the laceration. A, Local injection, B, Epinephrine 1 c.c.

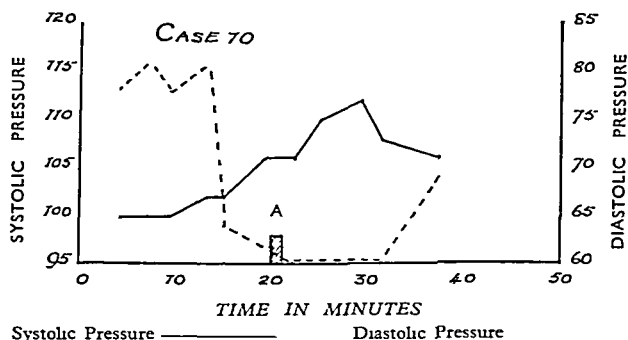


FIG. 482.—*Case 10*. F. M., male, aged 10. Admitted with fractured skull, fractured humerus, and first degree Pott's fracture. Some clinical shock. Local infiltration to anesthetize site of fracture and to allow passing of Kirschner's wire through olecranon was made by the subcutaneous injection of 65 c.c. of 1 per cent procaine with 7 min. of adrenaline. A, Local injection.

In *Fig. 481* the record resembles closely that obtained from brachial plexus blocks upon shocked patients. *Fig. 482* shows a slight rise in systolic pressure following local infiltration.

Figs. 483-485 illustrate the effect of local infiltration upon patients in whom no degree of shock was to be expected, and *Case 14* showed no clinical evidence of shock (Fig. 486).

On the whole it may be said that local tissue infiltration exerts the same effect upon the blood-pressure as does blocking the brachial plexus; and that the action is a depressor one produced by the local anæsthetic independent of the site of

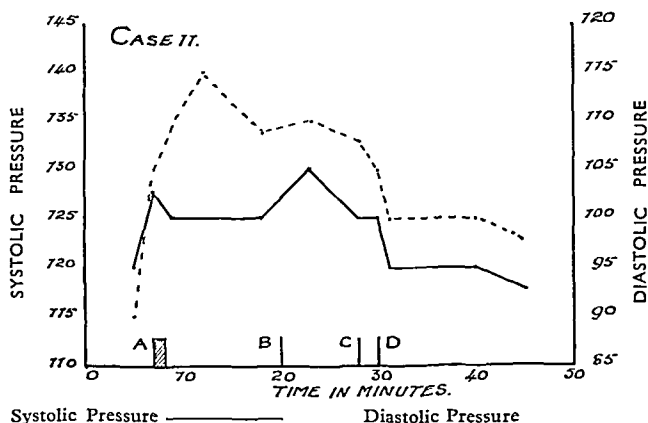


FIG. 483.—*Case 11.* W. H., male, aged 46. Laparotomy for inoperable carcinoma of the stomach. Infiltration of the abdominal wall and celiac ganglion block was made with 300 c.c. of $\frac{1}{2}$ per cent 'Novutox' and 30 min. of adrenaline. A, Local injection begun; B, Operation started; C, Parietal peritoneum injected; D, Celiac ganglion injected.

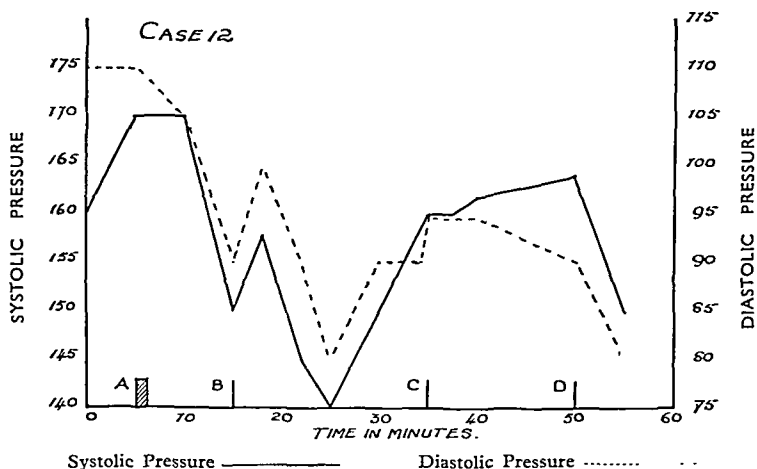


FIG. 484.—*Case 12.* F. W., male, aged 48. Laparotomy for pain following gastrojejunostomy (? jejunal ulcer). Abdominal wall infiltration and splanchnic block were made by injecting 330 c.c. of $\frac{1}{2}$ per cent 'Novutox' and 30 min. adrenaline. A, Local injection begun; B, Skin incision; Patient felt faint; C, Splanchnic block; D, Sewing up.

introduction. Further, whilst brachial plexus block is contra-indicated in traumatic lesions of the upper limb, local anæsthesia in general is unsuitable for shocked patients when large doses of anæsthetic are required. It must be stressed that the above findings neither substantiate nor invalidate the 'nerve theory' of traumatic shock. If plexus block has any effect upon traumatic shock, this is overshadowed by the depressor effect of the anæsthetic when given in doses sufficient to ensure

complete blocking of the plexus. This drop in blood-pressure may be controlled by ephedrine (*Case 2, Fig. 474*) or by the injection of large doses of adrenaline as is illustrated by *Case 15 (Fig. 487)*, where a dissection of the axillary glands was undertaken under local infiltration anæsthesia, using a disproportionately strong dose of adrenaline.

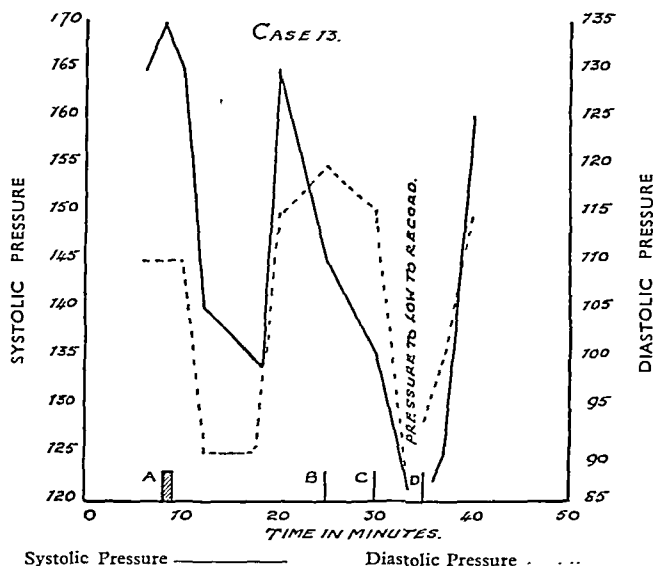


FIG. 485.—*Case 13.* G. H., male, aged 58. Laparotomy for inoperable leather-bottle stomach. Infiltration of lesser omentum with carcinoma precluded the possibility of injecting the celiac ganglion. Abdominal wall infiltration with 290 c.c. of $\frac{1}{2}$ per cent 'Novutox' and 30 min. adrenaline. A, Local injection begun; B, Skin incision; C, Attempt to inject celiac ganglion; D, Sewing up.

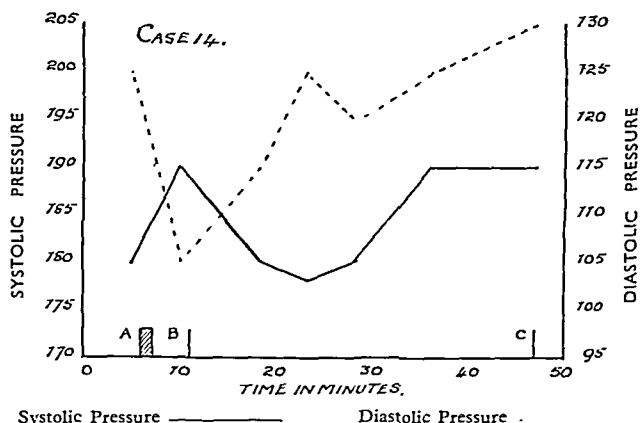


FIG. 486.—*Case 14.* M. B., female, aged 62. Admitted for strangulated umbilical hernia. Repaired by Mayo's method; operation involved tension upon transverse mesocolon. Local infiltration of abdominal wall by injection of 150 c.c. 1 per cent procaine and 20 min. adrenaline. A, Local injection begun; B, Operation begun; C, Abdomen closed.

By way of comparison, typical blood-pressure readings of a case of burns accompanied by moderate shock, and anesthetized with chloroform and ether, are shown in *Fig. 488*. Here the blood-pressure is fairly well maintained, as it was

in all such cases examined; and no anxiety was ever felt on behalf of patients in a moderately shocked condition under ether anaesthesia, although this method of anaesthesia is admittedly inferior to gas and oxygen in shocked patients.

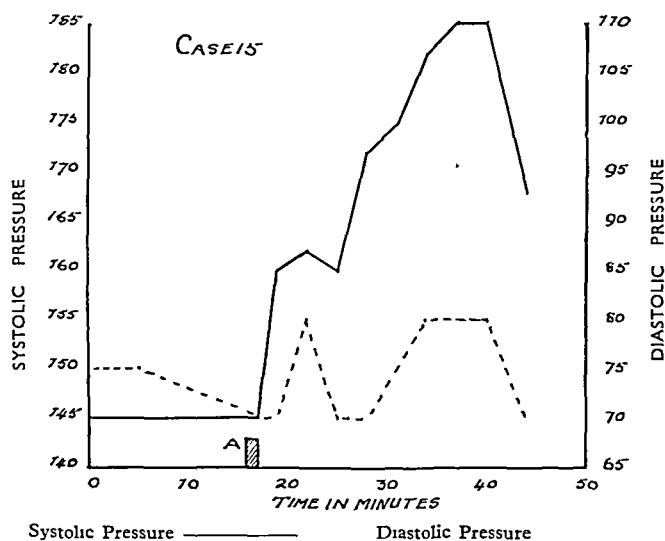


FIG. 487.—Case 15. G. L., male, aged 35. Biopsy on deep-seated gland in the axilla. Subcutaneous injection of 100 c.c. 1 per cent procaine and 25 min adrenaline. A, Local injection.

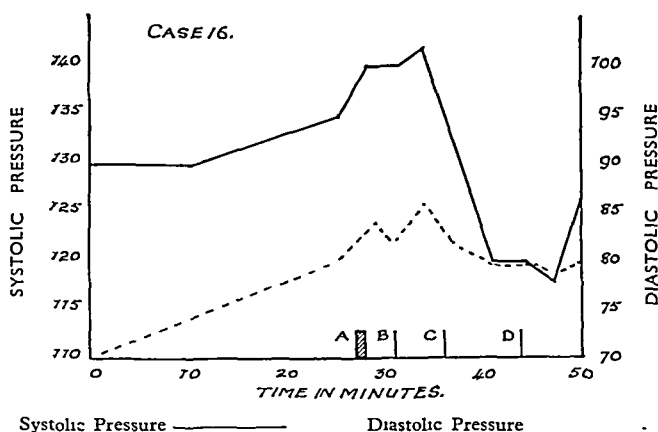


FIG. 488.—Case 16. E. H., male, aged 35. Second degree burns of forearm and hands—left, 100 sq. in., right, 144 sq. in. There was slight shock. Chloroform and ether anaesthesia. A, Chloroform and ether; B, Ether; C, Cleaning; D, Tanning.

SUMMARY

1. An investigation was carried out to determine the effect upon the blood-pressure of blocking the brachial plexus in patients suffering from shock due to trauma to the upper limb.

2. A series of controls was also examined in which blood-pressure readings were taken on patients receiving a brachial plexus block who were not clinically shocked and on patients receiving local infiltration into the tissues.

CONCLUSIONS

1. Brachial plexus block exerts no protective influence upon patients suffering from shock due to trauma of the upper limb.
2. Both brachial plexus block and local infiltration anaesthesia tend to cause a fall in blood-pressure, particularly in shocked patients.
3. This fall in blood-pressure is probably due to the local anaesthetic, irrespective of the site of introduction.
4. Inhalation anaesthesia is a more suitable anaesthetic for shocked patients.

My thanks are due to the staff of Guy's Hospital for allowing me to publish these findings upon their cases ; to Professor Grant, of the Clinical Research Unit, for his criticism and help ; and particularly to the house officers, dressers, and ward clerks, who, often in the small hours of the morning, have taken blood-pressure readings whilst the time-consuming performance of brachial plexus anaesthesia was being enacted.

REFERENCES

- ¹ FRASER and COWELL, *Med. Research Council Special Rep. Series*, No. 25, ii (1), 49.
- ² O'SHAUGHNESSY and SLOME, *Brit. Jour. Surg.*, 1935, lxxxvii, 589.

FIXATION OF THE HIP-JOINT BY MEANS OF AN EXTRA-ARTICULAR BONE-GRAFT: LATE RESULTS

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IN the year 1932 I described a method of fixation of the hip-joint by means of an extra-articular bone-graft.¹ Sufficient time has elapsed since the performance of my earlier operations to enable me to make certain observations which may be of interest to surgeons.

The main features of the operation are as follows. The skin and the fascia lata of the thigh are incised in the same line and the osseous insertion of the gluteus maximus muscle is divided a short distance from the bone. The musculo-cutaneous flap so formed is elevated enough to expose the tuberosity of the ischium, the femoral shaft, and the sciatic nerves (*Fig. 489*). A deep cleft is made in the tuberosity of the ischium with a broad chisel, and a trap-door opening is cut in the postero-medial aspect of the shaft of the femur a little below the level of the lesser trochanter. A stout free bone-graft of the requisite length is cut from the tibia and inserted as shown in *Figs. 490 and 491*. Splinting is of importance, and is described in the original paper.

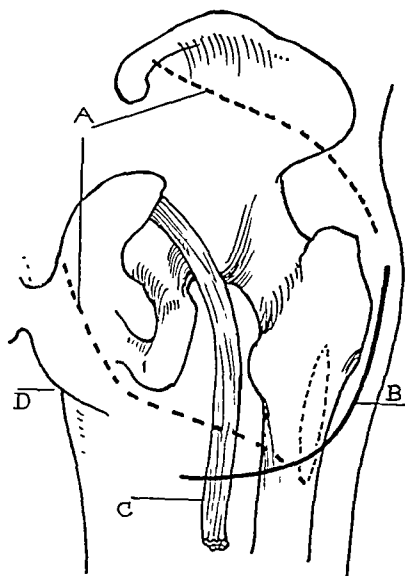


FIG. 489.—Showing line of incision and how it skirts the insertion of the gluteus maximus into the fascia lata above and the femur below, and crosses the middle line of the thigh below the level of the gluteal sulcus. The limits of the gluteus maximus are represented by the broken line, the osseous insertion by the area enclosed in the interrupted line on the femoral shaft. A, Outline of gluteus maximus; B, Line of incision; C, Sciatic nerve; D, Gluteal sulcus.

I have performed the operation 8 times for the fixation of tuberculous hip-joints. There were no post-operative troubles of any importance and no immediate infection or subsequent breaking-down of the wounds. This is one advantage of working in normal tissues well removed from the tuberculous area. Three of the patients had sinuses discharging on the lateral and anterior aspects of the hip-joint at the time of operation, whilst in another (*Case 1*) there were scars of healed sinuses. Grafting of bone in the region of the femoral neck and acetabulum in these cases was inadvisable. Some months after operation in *Case 1* an abscess developed in the region of the great trochanter and discharged through a scar which marked the site of a sinus which

had healed some years previously. Had the graft been inserted on the lateral aspect of the hip-joint, the result in all probability would have been disastrous. In two patients the sinuses have since healed.

In 7 of the patients the graft united satisfactorily both with the femoral shaft and the ischial tuberosity. In one case the graft became displaced from the cleft made in the ischial tuberosity soon after operation by some ill-advised movement of the limb, but united strongly with the femur. Absolute fixation of the hip-joint was attained in all but the last case.

In the years after operation, increase in thickness and strength of the grafts has been very striking, and in some of the cases unexpectedly pronounced. In several instances the radiographic appearances are sufficient to show that the grafts, besides acting as distance bars or struts preventing adduction and movement at the hip-joint, are transmitting a large proportion of the body weight to the femoral shafts. In *Case 1* the graft appears to be much stronger than the femoral neck (*Figs. 492, 493*).

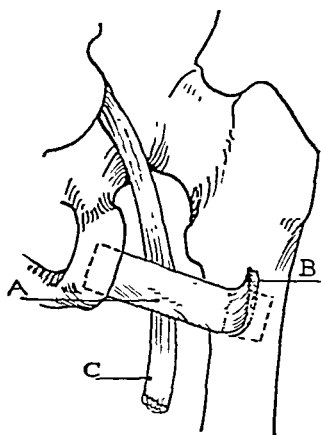


FIG. 490.—Diagrammatic representation of graft in position, one end being inserted into a cleft in the tuberosity of the ischium, the other into the medullary cavity of the femur through a trap-door opening. The trap-door, hinged on the periosteum at its lateral margin, is shown open. A, Graft in position; B, Trap-door open; C, Sciatic nerve.

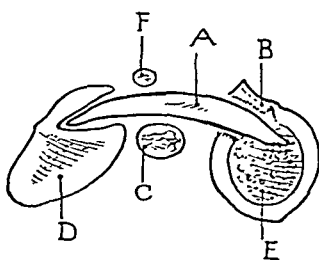


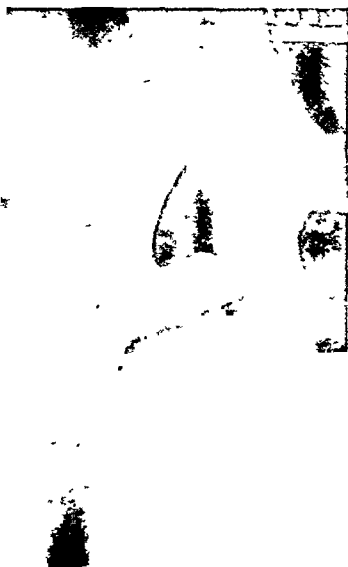
FIG. 491.—Diagrammatic representation of horizontal section through the graft in position, showing how it arches over the great sciatic nerve and the manner in which the ends are cut to a wedge shape to facilitate introduction. Both ends are firmly gripped and cannot get out of position. The trap-door in the femur is shown replaced. The sciatic nerves are shown separated by the graft. A, Graft; B, Trap-door closed; C, Great sciatic nerve; D, Ischial tuberosity; E, Femur; F, Lesser sciatic nerve. (*Figs. 489-491 are reproduced by the kind permission of Sir Hugh Devine and 'The Australian and New Zealand Journal of Surgery.'*)

No increase in deformity has occurred in any case. In some, the position of the hip-joint at the time of operation was not ideal. In no instance has there been any further destruction of bones entering into the formation of the hip-joint, and in all but one the tuberculous process seems to have died out. Bony union at the hip-joint appears to have taken place in four of the cases (*Figs. 495, 498, 499*). This would seem to be directly due to the absolute fixation of the hip-joint effected by the graft.

Fracture of the graft occurred in one case, about eighteen months after operation. The graft in question was a broad flat piece of bone cut obliquely from the subcutaneous surface of the tibia (*Fig. 496*). At its centre it was thin, and at this point fracture took place. The patient, a woman, aged 34 years at the time of operation, had been confined to bed for about eight years with multiple tuberculous lesions, and all her bones were very atrophic. Skiagrams taken at intervals after the time of fracture show that the fragments have increased in size and density and that there has been some attempt at union, which, however, is not certainly



FIGS. 492, 493.—Skiagrams of hip-joint in Case 1, taken respectively 5 and 54 months after operation. Note the tremendous hypertrophy of the graft, and the relative fragility of the neck and head of the femur in the later skiagram.



FIGS. 494, 495.—Skiagrams of hip-joint in Case 2, taken respectively 1 and 60 months after operation. Note the changes in the graft, and osseous ankylosis at the hip-joint.



FIGS. 496, 497.—Skiagraphs of the hip-joint in *Case 3*, taken respectively 7 and 59 months after operation. Fracture occurred about eighteen months after operation. The later skiagraph shows that there has been some attempt at union, which is possibly bony.



FIG. 498.—Skiagraph of hip-joint in *Case 7*, taken 46 months after operation. Probable bony ankylosis.



FIG. 499.—Skiagraph of hip-joint in *Case 8*, taken 33 months after operation. Note the apparent extension of the medullary cavity of the femoral shaft into the graft, and the bony ankylosis at the hip-joint.

bony. There is no movement clinically appreciable at the hip-joint (*Figs. 496, 497*).

At present (April, 1936) all but one of the patients are up and about. The man in whom the graft failed to unite with the ischium is confined to bed with extensive pulmonary tuberculosis, although the hip disease is quiescent. A second man, after being up for a period, developed an intrapelvic abscess which was eventually opened by Mr. Balcombe Quick. Sinuses are still discharging on the anterior aspect of the joint, although skiagrams show that the graft has united firmly at both ends and fixation of the hip is quite satisfactory. The patient feels well on the whole, but at times rather ill when blockage of sinuses occurs. The remaining six patients are very well, having dispensed with all artificial aids to walking. Several have returned to their former occupations.

The details of the cases are summarized in the following Table.

TABLE GIVING BRIEF DETAILS OF CASE NOTES OF EIGHT PATIENTS OPERATED UPON PRIOR TO MAY, 1936

CASE NO.	SEX AND AGE IN YEARS	DATE OF OPERATION	PRESENT STATE, MAY, 1936		
			General Health	Hip-joint	Graft
1	M 18	10/10/30	Excellent, working	Firm fixation. Painless. Sinus healed	Greatly hypertrophied. Union excellent
2	F 20	14/3/31	Excellent, working	Bony ankylosis. Painless	Greatly hypertrophied. Union excellent
3	F 34	11/4/31	Good	Firm fixation. Painless	Fractured at 18 months. ? Re-united
4	M 31	18/3/32	Fair. Secondary infection of hip with sinuses	Firmly fixed. Painless. Several sinuses discharging	Good union. Moderate hypertrophy
5	M 24	29/3/32	Pulmonary Tb. Confined to bed	Slight movement. Painless	Failed to unite with ischium
6	F 35	8/6/32	Excellent	Bony ankylosis. Painless	Good union and hypertrophy
7	M 18	8/6/32	Excellent	Bony ankylosis. Painless. Sinus discharging	Good union and hypertrophy
8	F 15	9/5/33	Excellent	Bony ankylosis. Painless. Sinus healed	Good union and hypertrophy

REFERENCE

- ¹ TRUMBLE, H. C., "A Method of Fixation of the Hip-joint by means of an Extra-articular Bone-Graft", *Australian and N.Z. Jour. Surg.*, 1931-2, 1, 413.

THE TREATMENT OF FRACTURED PATELLA BY EXCISION. A STUDY OF MORPHOLOGY AND FUNCTION

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MORPHOLOGY OF THE PATELLA

THE patella is classed as a sesamoid bone, but as far as this particular bone is concerned, it is the view of the author that phylogeny and phylogeny alone is responsible both for its presence and development, and that there is no evidence that its development is a response to a functional need or that the bone serves any useful function when it is formed. It is, in other words, an integral part of the skeleton phylogenetically inherited. Actually Bernays,¹ and later Kazzender,² have shown that the patella is neither situated nor developed in the tendon of the

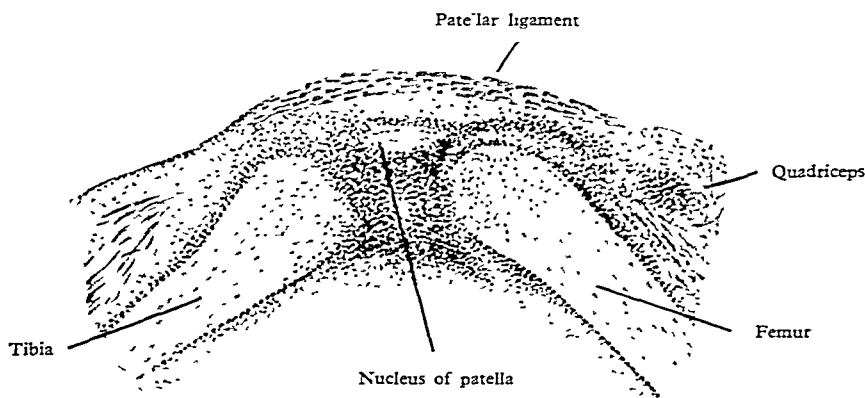


FIG. 500.—Drawing from microphotograph of section through the knee-joint of an embryo 50 mm. long. The nucleus which is to become the patella is already well formed, and lies behind a stratum of cells, well differentiated, which are to become the ligamentum patellæ.

quadriceps, but quite independently and behind the tendon. It is true that the latter subsequently obtains attachments to it, but the two are really quite distinct (Fig. 500).

Comparative Anatomy.—The functional activity of the quadriceps muscle in man increases on the whole from birth until adult life is reached, and the relative size of the patella, if governed by function, should be proportionately larger in the adult. Actually, it is relatively smaller. The same holds in the case of the adult lion and bear. There is also no relation between the depth of the patellar fossa of the femur and the size of the patella or the activity of the animal. In short, there would seem to be no indication that in animals function influences the development and growth of the patella. Thus in sloths, moles, and echidnæ, all slowly moving animals, the bone is massive and proportionately well developed,

while in the fox, deer, and leopard, all rapidly moving animals, the bone is proportionately small. Finally, in the kangaroo, in which the quadriceps is developed enormously in conformation to the animal's mode of progression, the bone is completely absent. De Vries^{3, 4} has pointed out that in animals of the older orders, and in animals that are now extinct, the patella is relatively larger and more important than it is in the younger members of the same family. From this and other observations she deduced that the bone is in the process of reduction in much the same way as the fibula is, a strong point in favour of phylogeny as the determining factor for its presence.

ROLE OF THE PATELLA IN THE MECHANISM OF THE KNEE-JOINT

Lickey has pointed out that the bone forms a more suitable pulley for movement round the condyles of the femur than the tendon itself, for the upper end of the patella is kept in a plane well in front of the axis of flexion and extension, thus allowing the quadriceps to act to greater advantage in producing extension. He suggests that the patella supplies the quadriceps muscle with a lever of the first order, acted on by a force at each end, with a fulcrum at a varying distance between the two. The forces acting at the ends are each subject to variations during the movement of flexion and extension, and are dependent on the angles made by the line of the resulting forces of the quadriceps with the long axis of the femur, and that made by the ligamentum patellæ with the long axis of the tibia; and the angles made by the line of the resultant forces of the quadriceps with the patella, and the angle made by the ligamentum patellæ and the patella.

All these angles are constantly varying during extension, but the variation is so slight that for practical purposes the forces acting on each end of the bone may be regarded as constant. Any variation of the force must then be due to alteration in the position of the fulcrum on the back of the patella. The position of the fulcrum is constantly altering; thus, when the tibia is moved, the force acts upon the upper segment of the patella, and the fulcrum in the extended position is on the lower facets of the patella surface, close to the lower end of the patella. In the position of full flexion it is nearer the upper end. The force, therefore, in the position of extension acts to much greater advantage than in the flexed position, or, in other words, the turning movement of the force increases whilst that of the resistance diminishes in the transmission from flexion to extension. Now, as the velocity is proportional to the force causing motion, the velocity will be greatest at the end of extension. When the tibia is fixed, the conditions obtaining are reversed. The force is now most effective in the flexed, and least effective in the extended, position, and the velocity therefore will diminish as extension is effected. With the tibia movable, the act of extension produces the act of kicking, and at the moment of impact the greatest momentum is required. The mass being a fixed quantity, this is obtained by increasing the velocity up to the end of the act by altering the fulcrum. When the tibia is fixed, the movement of extension is required for the effort of raising a considerable portion of the weight of the body, so that in this case the greatest amount of force is necessary at the beginning of the action. A diminution of the force, and with it the velocity, is essential at the end of the act in order that the body be not overbalanced.

This explanation of the mechanics of the knee-joint is probably correct. How much the leverage action and alteration of fulcrum in the knee-joint is an advantage to the individual is in question. Comparative anatomy, as already mentioned, would tend to show that in animals, at least, any such advantage is unimportant. The lever must at least be a very small one, and any mechanical advantage which might be gained by it is more than counteracted by the interference with rate and loss of smoothness of motion, associated with the alteration in the position of the fulcrum. In man, the active range of movement at the knee-joint in walking and running is just short of extension, a position in which the force is least efficient as a weight-raising mechanism.

The patella has been adapted to play a part in the movements of the knee-joint, but it was not designed for this purpose; and although, theoretically, its presence should enhance the power of the quadriceps muscle, in practice there is considerable doubt as to its mechanical value in assisting movements at that joint. Rather is there evidence that it has become imperfectly adapted to the mechanism of the joint, and thus has a deterrent action on a machine for which it was never designed.

EXPERIMENTAL INVESTIGATION OF THE IMPORTANCE OF THE PATELLA IN MOVEMENTS AT THE KNEE-JOINT

In order that more light might be thrown on the part which the patella plays in movements of the knee-joint, certain experiments have been performed. For this purpose, a number of knee-joints in the fresh state were obtained from the

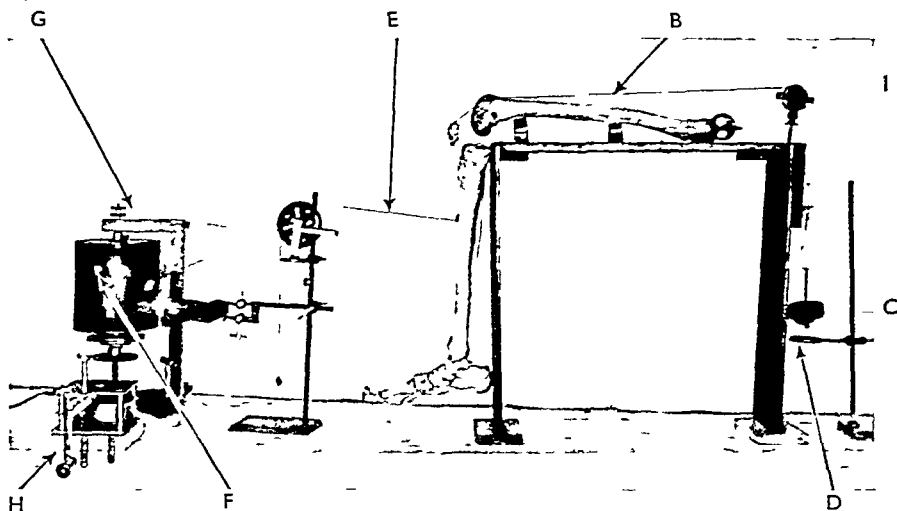


FIG. 501.—Apparatus for determining the influence of the patella on the movements of the knee-joint. For the knee-joint freshly excised from the cadaver, a disarticulated skeleton of the lower limb has been substituted in the photograph for convenience. B, Lead attached to end of quadriceps tendon; C, Weight attached to end of cord; D, Load-releasing mechanism; E, Lead attached to tibial spine; F, Lever writing on revolving drum; G, Revolving drum; H, Motor; I, Peg used as locking device.

post-mortem room and freed from all muscular attachments with the exception of a short length of quadriceps tendon, which was left attached to the patella. The specimen was then mounted on a stand (*Fig. 501*). A cord, B, was attached to the

stump of the quadriceps tendon, passing over a pulley with a weight, C, attached to the end. D is a load-releasing mechanism. A second weighted cord, E, is attached to the anterior surface of the tibia just below the tibial tuberosity, passing in turn over a reducing pulley, and then to the lever F, writing on a revolving drum, G; H is a motor connected with the drum. A weight sufficient to lift the tibia just short of the horizontal was attached to the cord B. The peg I was then withdrawn and the tibia rose, tracing as it did so a curve on the recording drum. When the movement had ceased the weight was released by the weight-relieving mechanism, D, and the tibia fell to its original position. In later experiments this apparatus was modified and somewhat simplified. The weight-relieving mechanism was found to be unnecessary. Tracings were first obtained of movements of the excised joints with the patella in situ. The patella was then carefully removed through a cruciate incision in the quadriceps tendon, after which the flaps were carefully replaced and sewn together again. The experiment was then repeated,

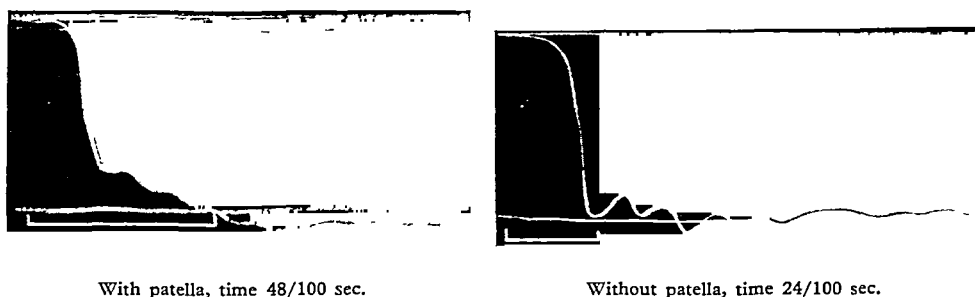


FIG. 502.—Tracing obtained for movement through a right angle, using the apparatus as illustrated in Fig. 501. Weight X attached to quadriceps.

and tracings were obtained, this time without the patella. Finally, the rent made by removing the patella was left widely open, and tracings were obtained of movement in this state.

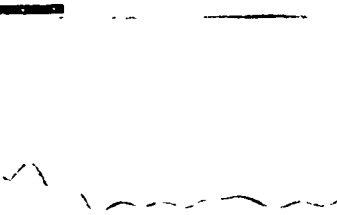
Movements through a right angle were first recorded, and then through the complete range of movement. A different stand was used in each case. The weight producing the movement was also altered so that the response to variations in the force might be noted.

With a weight X attached to the quadriceps tendon and with the patella present, the tibia was found to move from the vertical position to just short of the horizontal. The movement took place at a uniform rate throughout the first two-thirds of the movement, and then the rate of movement slowed off until the resting position was reached. The time taken for the whole movement was 48/100 of a second (Fig. 502).

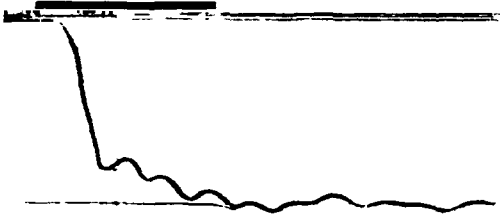
When the patella was absent and the gap in the patellar ligament had been closed, the movement was more uniform; there was no slowing towards the end of the movement, and the time taken for the movement was 24/100 of a second—approximately half the time when the bone was present (Fig. 502).

When a smaller weight, *Y*, was used as the force, the time taken for the right angle range of movement with the patella present was 38/100 of a second in one instance, and 42/100 of a second in another. In each case the movement was at first rapid and subsequently tailed off (*Fig. 503*).

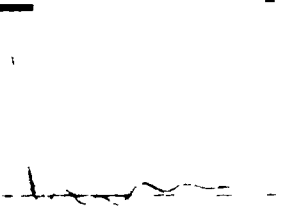
When the patella was absent, and the gap made by its removal was left unsewn, the time taken over the whole movement was 22/100 and 24/100 of a second in two instances. The movement was uniform, and there was no tailing off towards the end. The actual range of movement was the same in each case (*Fig. 503*).




With patella, time 38/100 sec.




With patella, time 42/100 sec.



Without patella, time 24/100 sec.



Without patella, time 22/100 sec.



Time marker, 50 per sec.

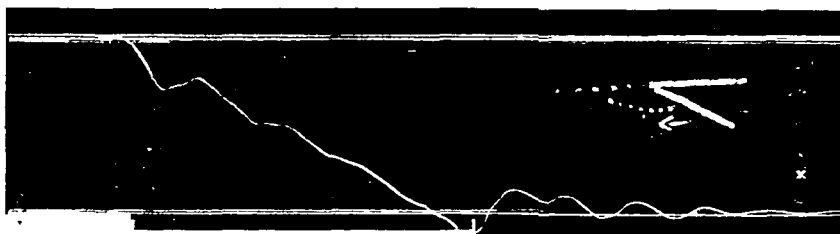
FIG. 503.—Tracing of movement through a right angle, with smaller weight, *Y*.

When a weight *X* was used and the complete range of movement (from acute flexion to full extension) was recorded with the patella present, the time taken was 88/100 of a second. The movement commenced sharply, but very rapidly slowed down to a uniform rate, which was maintained throughout; thus there was no tailing off as in the previous experiment when the movement was through a right angle only (*Fig. 504*).

When the movement was recorded without the patella and with the gap in the quadriceps sewn, the time was approximately 76/100 of a second, 12/100 of a second less than the time taken when the bone was present. The movement commenced at a quicker rate, and then very rapidly slowed down to a uniform rate, which was maintained throughout. The smaller acceleration of movement at the commencement is probably due to the taking in of slack, thereby imparting to the system an initial thrust (*Fig. 504*).



With patella, time 88/100 sec.

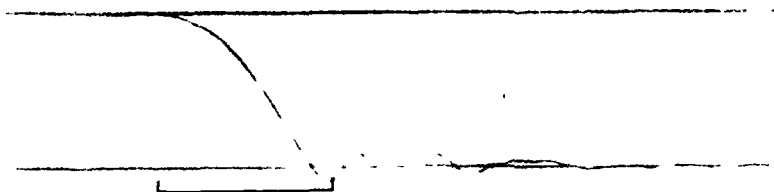


Without patella, time 76/100 sec.

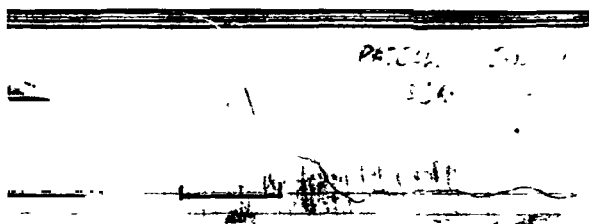


Time marker, 50 per sec.

FIG. 504.—Tracing obtained with complete range of movement and with weight, X.



With patella, time 40/100 sec.



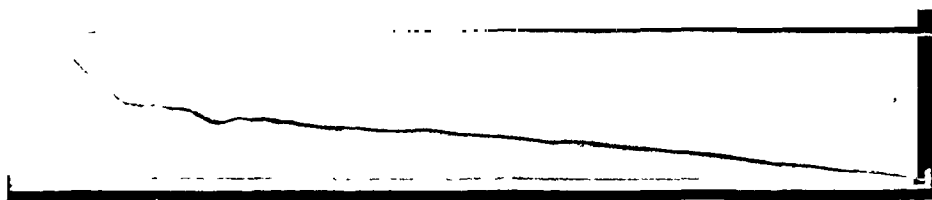
Without patella, time 22/100 sec



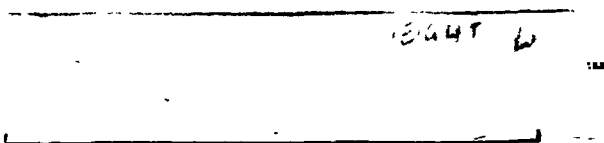
Time marker, 50 per sec.

FIG. 505.—Movement through right angle only, weight X attached to quadriceps. A repeat experiment (cf. Fig. 502).

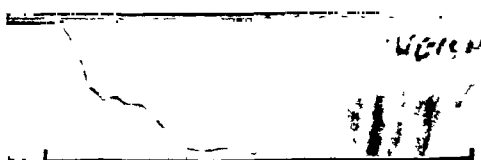
A repetition of the experiment with a weight *X* and the movement through a right angle only, with the patella present, gave a time of 40/100 of a second for the movement. When the patella was absent, the time was 22/100 of a second (*Fig. 505*). In this tracing the usual lag towards the end of the movement, when the patella was present, was not a feature—in fact, the movement towards the end was, if anything, more rapid.



With patella, time 56/100 sec.



Without patella, tendon not sewn, time 22/100 sec.



Without patella, tendon sewn, time 1 sec.



Time marker, 50 per sec.

FIG. 506.—Movement through complete range, with smaller weight, *Y*. The functional result is little altered when the patellar tendon is left unsewn.

With a weight *Y* as the force acting through the complete range of movement, and with the patella present, the time taken was 56/100 of a second. With the patella absent, and the gap in the patellar ligament not sewn, the time was 22/100 of a second, and 1 second when the ligament was sewn (*Fig. 506*). The amplitude of the contraction was the same in each case. It would thus seem that, provided the lateral expansions of the patellar ligament are intact, there is very little functional difference whether the gap left after the removal of the patella is sewn up or left open.

REMOVAL OF THE PATELLA FOR SIMPLE TRANSVERSE FRACTURES, AND ITS EFFECT ON THE FUNCTION OF THE KNEE-JOINT

Tuberculosis of the bone, compound stellate fractures, and, during the War, injury from gunshot wounds, are perhaps the three most common conditions for which the patella has been removed. With a few exceptions the results have been disappointing and the subsequent function of the knee-joint impaired. This is in all probability due to the fact that the disease or damage was not confined to the bone but involved the soft tissues as well.



FIG 507—Dynamometer for measuring the strength of the quadriceps muscle. Attached to the patient's ankle is a strap, A, passing in turn to the graduated recording spring, B, attached to the lever, C. The foot of the subject is raised from the ground and the toe is held against the wooden block, D. The lever is pulled backwards and the force necessary to just pull the toe away from the wooden block is read off on the graduated spring scale.

Effect on the Strength of the Leg of the Removal of the Patella.—By means of the simple apparatus shown in *Fig. 507*, the strength of the knee extension can be measured.

In ten cases where measurements were taken, it was found by independent observers that, with only one exception, the leg from which the patella had been removed was actually stronger than its fellow, in the matter of resisting flexion, by an amount varying from 1 to 5 lb.

The Present Series of Cases.—During the past seven years I have operated upon and removed the patella from 30 cases of simple fracture of the patella (eight women and twenty-two men). In one instance both bones were broken at different times. In one case the excision was done after wiring had been done twice and had resulted in a stiff knee. In one case it was done after the patella had been fractured in manipulating a stiff knee after a comminuted fracture of the lower third of the femur. Two facts stand out very clearly as the result of all these cases.

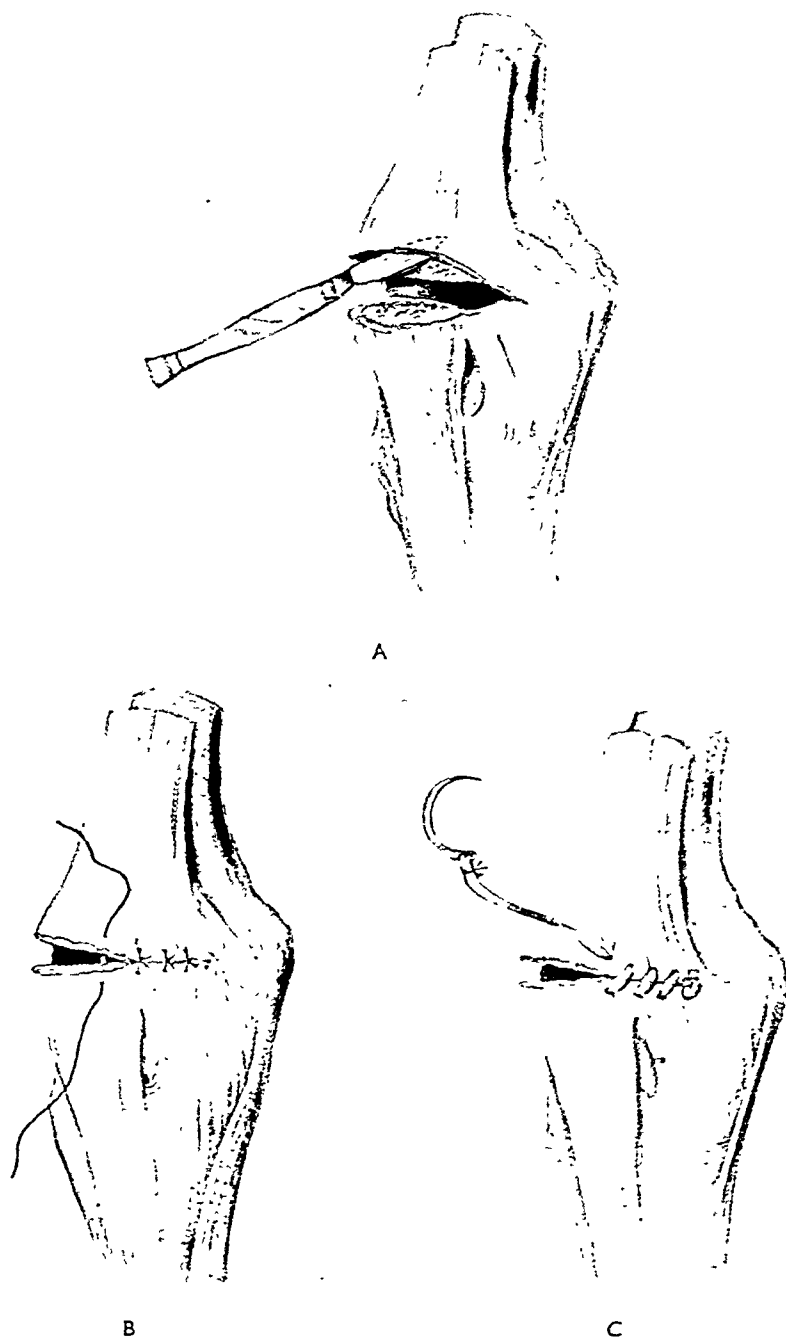


FIG. 508.—Showing technique of the operation. A, The fractured portions of the patella are shelled out of the tendon of the patella and the fibrous expansions of the quadriceps muscle. B, The transverse gap is then closed with interrupted sutures of silk. Great care is taken to sew the lateral expansions. C, As an alternative to the silk sutures, strips of fascia lata may be used.

First, the rapid and smooth recovery, which usually only takes two or three weeks. Second, the very complete recovery of function, so that labouring men can return to full work—e.g., ladder climbing—within a month or six weeks of the operation.

Operation.—After the accident the limb is firmly bandaged over wool to control swelling. After forty-eight hours the patella is removed through a vertical incision, being easily shelled out of its aponeurotic covering (*Fig. 508, A*). The quadriceps tendon is then carefully sewn together either by a fascial strip (*Fig. 508, C*), or by interrupted silk sutures (*Fig. 508, B*). A firm domette bandage is applied over an ample layer of cotton-wool. This is the only splint. After two days the patient is allowed up. After ten days the wool and bandage are taken off and the stitches removed. The patient is able to return to work in two to six weeks from the date of operation, according to the nature of his occupation.

Some typical cases are illustrated in *Figs. 509–518*.



A



B



C

FIG. 509.—Man of 22. Fracture of right patella (A). After excision of fragments he was able to return to work in sixteen days (B). Three years later he broke the left patella. Fragments excised, returned to work in three weeks (C).



FIG. 510.—Three years previously, this patient, a male aged 51, was operated upon for a stellate fracture of the left patella with considerable separation of the fragments. The fracture was compound. The bone was removed and the gap sutured with fascia obtained from the thigh. The left quadriceps resists a pull of 9 lb. more than the right, and the patient, when working, notices no difference in the two limbs.

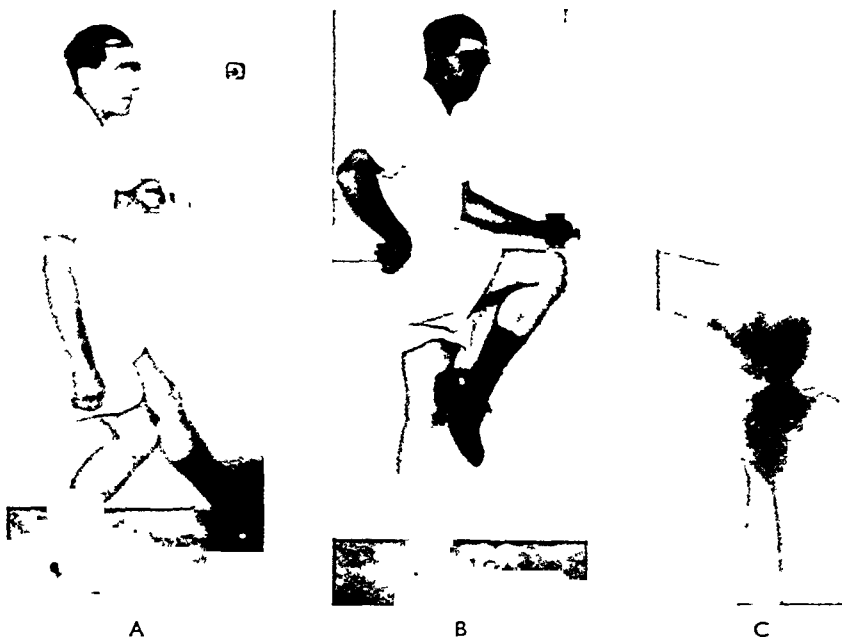


FIG. 511.—Man of 34. Transverse fracture of right patella. Fragments removed, gap repaired by silk sutures. He walked two days later, and in three weeks returned to work. Two years later he was unable to distinguish any difference between one leg and the other. A, Body weight supported on the semi-flexed knee; B, Full flexion of knee; C, Radiograph of knee.



FIG. 512.—Man of 23. Transverse fracture of left patella. Excision and suture. Returned to work in one month. Photographs show functional result.

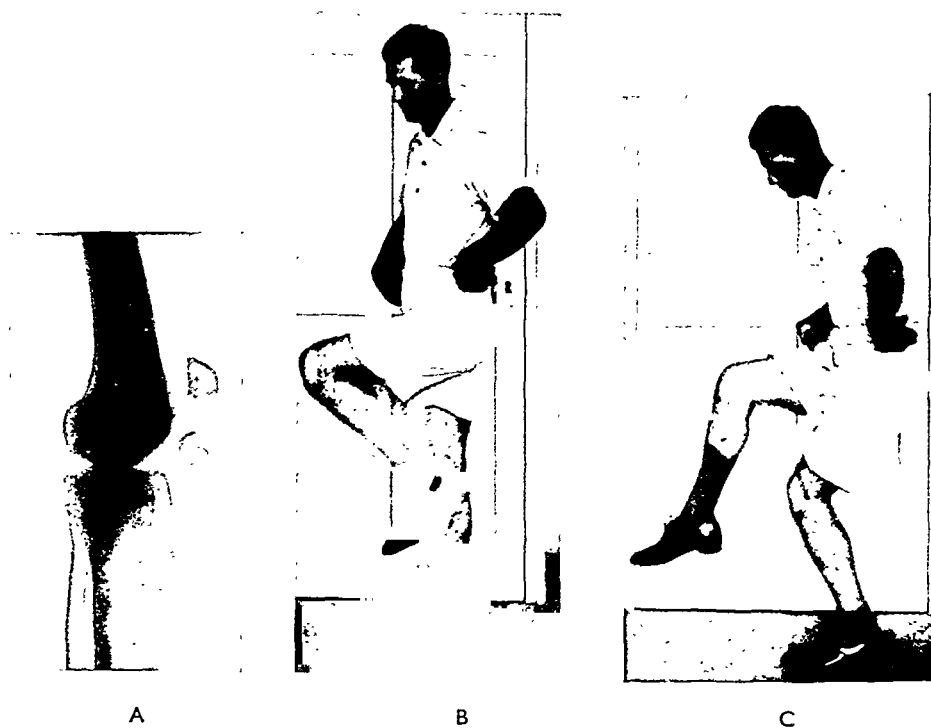


FIG. 513.—Window-cleaner aged 26. Transverse fracture of right patella (A). B and C show functional result one year later. He can do ladder work all day and one leg is as good as the other.

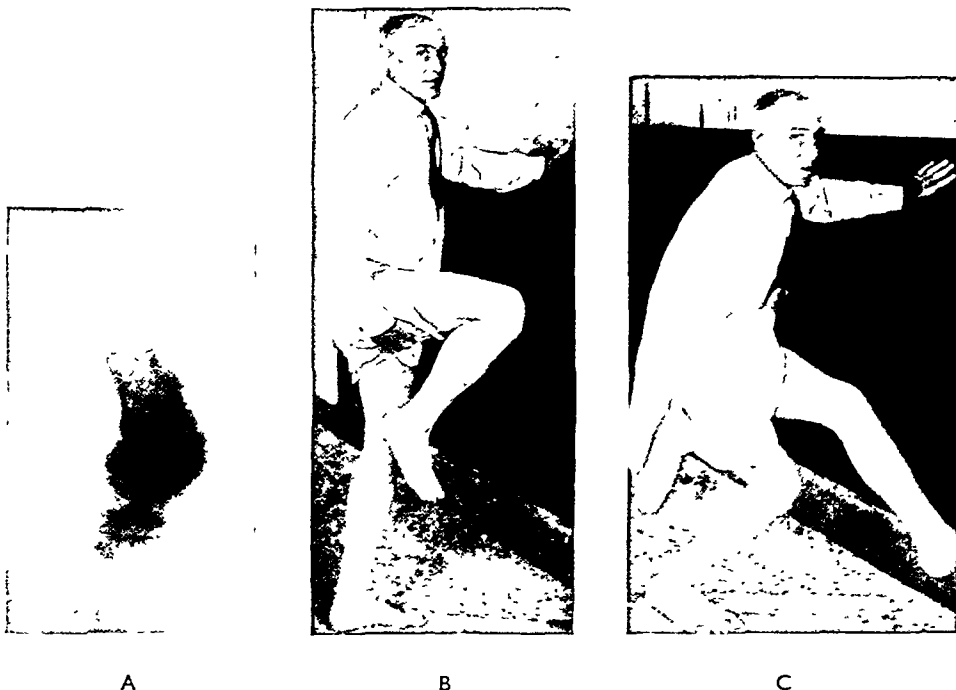


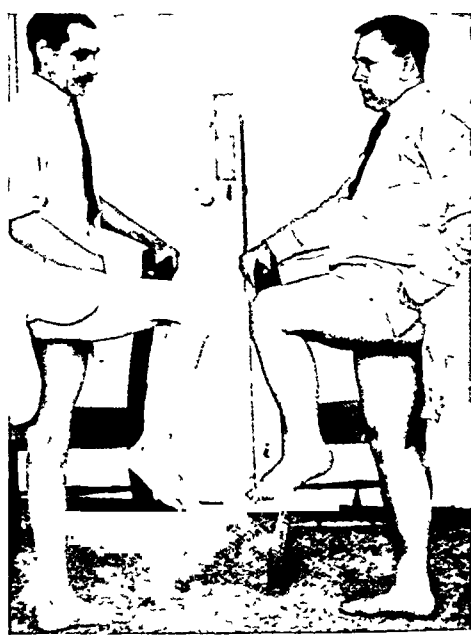
FIG. 514.—Labourer aged 56. Transverse fracture of right patella. Excision of fragments and suture (five years ago). A, Radiograph showing a few bony fragments in site of patella; B, Full flexion; C, Standing on the bent knee. He thinks that the right knee is stronger than the left.



FIG. 515.—Man aged 22. He had had a comminuted fracture of the lower end of the right femur and this had been plated. The plate had been removed, and for some time he wore a calliper. The knee was stiff. Manipulation under an anæsthetic caused fracture of the patella. The patellar fragments were excised. Eight weeks later he returned to work. After three years he had perfect function.



FIG. 516.—Man of 57. Transverse fracture of left patella. Excision. Returned to work within four weeks. Perfect function.



FIGS. 517, 518.—*Left Patient:* Man of 32. Transverse fracture of left patella. Excision and suture. Discharged after eleven days. Two years later he could not distinguish any difference between the two legs. *Right Patient:* Man of 45. Transverse fracture of right patella. Excision and suture. One year later function was perfect.

CONCLUSIONS

The patella is an integral part of the skeleton phylogenically inherited, and function plays no part either in its formation or its growth. In man it subserves no important function. It is a morphological remnant which is tending to undergo reduction and to disappear. It has become modified to take part in movements of the knee-joint, but its presence is incidental and is a deterrent rather than an aid to these movements. Experimental and other evidence has been advanced to show that in its absence the efficiency of the knee-joint is, if anything, increased, both as regards the rapidity of movement and power.

REFERENCES

- ¹BERNAYS, A., "Die Entwicklungsgeschichte des Kniegelenkes des Menschen mit Bemerkungen über die Gelenke im allgemeinen", *Morphol. Jahrb.* 1878, iv.
- ²KAZZENDER, J., "Ueber die Entwicklung des Kniegelenkes", *Arch. f. Anat. u. Entwickl.*, 1894.
- ³VRIESE, BERTHA DE, "Zur Anatomie der Patella", *Verh. d. anat. Gesellsch. a. d. XXII. Vers.*, 1908. Berlin.
- ⁴VRIESE, BERTHA DE, "Anatomy of Patella", *Bull. de l'Acad. de Méd. de Belgique*, 1909; *Anat. Anzeiger Ergänzungsheft*, 1908; *Ann. et Mém. Soc. d'Anthropol. de Paris*, Series 6, iv. 1913.
- ⁵HESELWANDER, A., *Zeits. f. Morphol. u. Anthropol.* 1903, v; *Ibid.*, 1909, xii; *Anat. Anzeiger*, 1906, xxxii.
- ⁶BROOKE, R., *Proc. Roy. Soc. Med. (Orthop. Sect.)* 1937, xxx, 3.

A NOTE ON THE EXTENSION APPARATUS OF THE KNEE-JOINT

BY ERNEST W. HEY GROVES, BRISTOL

WHEN I first read Mr. Brooke's paper my reaction was one of frank incredulity. I asked if he would show me some of his cases, and he very promptly acceded to my request. In December he showed me eight of them, two women and six men, and I found that his claim of having restored these people to full functional activity by excision of the patella was fully justified. It was impossible in any case to detect any difference between the functional use of the injured and the uninjured leg. If the knee were covered by a bandage one could not guess which had been the damaged limb. I went away convinced, but still mystified as to how the mechanism of the extension of the knee could be so perfectly carried out in the absence of the patella.

I sought the help of Professor Whitnall, in the Anatomical Department of Bristol University. He kindly made two preparations for me which fully explain the matter. In one (*Fig. 519*) a longitudinal section of the knee-joint shows that the quadriceps tendon merely passes over the patella to become continuous with the patellar ligament below. The upper and lower margins of the patella are covered with fat and give no attachment to any ligamentous fibres. The second preparation (*Fig. 520*) shows a front view of the knee-joint after the patella has been excised. Pulling on the quadriceps tendon still produces extension of the knee. It is evident that quite apart from those fibres of the quadriceps tendon which pass

over the patella to the ligament below, there are ample fibres of the tendon present at each side of the patella to carry on continuity. Those on the medial side go partly to the patellar ligament and partly to the medial tuberosity of the tibia. Those on the lateral side go partly to the patellar tendon and partly to join the iliotibial band,

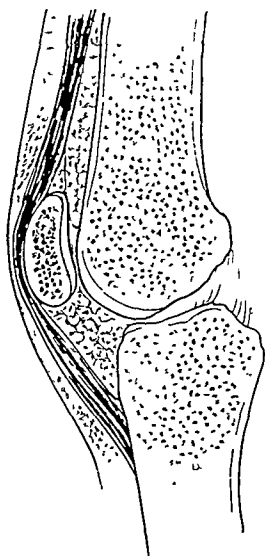


FIG. 519.—Longitudinal section of knee-joint, showing quadriceps tendon passing over patella.

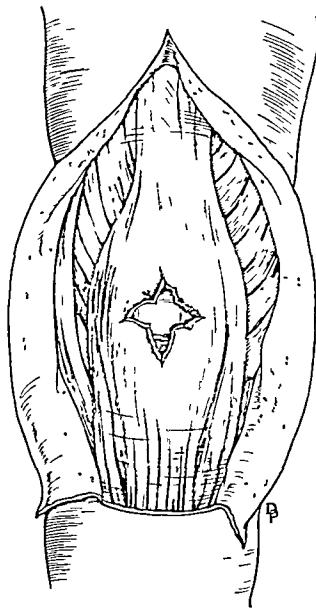


FIG. 520.—Anterior view of quadriceps tendon after removal of patella.

inserted into the lateral tuberosity. In every transverse fracture of the patella these lateral expansions of the quadriceps tendon are torn, and it is the suture of these lateral expansions which is the most important part of the operation of repair. It is clear that when the fragments of the fractured patella are removed, a much more close and firm repair can be made of the torn tendon.

SOME REFLECTIONS ON GASTROSTOMY

By E. S. J. KING

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IN the great majority of cases gastrostomy is performed merely as a palliative procedure, e.g., to maintain the nutrition of a patient with inoperable carcinoma of the œsophagus. Possibly for this reason the accounts of the operation given in text-books are inadequate and sometimes misleading. Even in the accounts where technical descriptions are complete, there is no statement of the principles involved. Indeed, usually a few methods only, and often the less important ones, are discussed.

A more serious omission is that of the contra-indications to the operation. In the literature the statement appears that certain forms of gastrostomy will never give post-operative trouble, e.g., leakage. Such statements indicate a lack of appreciation of even the small amount of available information concerning the physiology of the stomach, and disregard the many unsolved problems.

In this paper it is proposed to discuss the question from the point of view of difficulties that have arisen in the writer's experience of 50 cases. Gastrostomy becomes a very important procedure when one attempts by radical means the treatment of carcinoma of the œsophagus, and indeed is important in the treatment of any œsophageal condition where obstruction is present.

HISTORICAL

The observations of Beaumont³ (1825-1831, published 1833) on the stomach of a Canadian trapper, Alexis St. Martin, in whom a gastric fistula followed a gunshot wound, make this the most famous 'gastrostomy' in medical history.

A few years later (1837-1846) three surgeons—Egebert¹⁰ of Christiania, Watson⁴⁰ of New York, and Sédillot³¹ of Strasbourg—independently advocated gastrostomy as a means of feeding patients who had an impermeable stricture of the œsophagus. These suggestions apparently were inspired both by the records of Beaumont³ and by European reports of a similar though less complete nature. Attempts to produce a fistula were first made by Sédillot³¹, Fenger¹¹, and Maury²⁶, and these were followed by a number of others, but they all ended fatally.

The first successful gastrostomy was performed by Sydney Jones, at St. Thomas's Hospital, in 1875.²² Verneuil,³⁹ of Paris, had a successful case the next year. Following this many cases were operated on, with a mortality ranging from 80 to 20 per cent. When one considers the dangers and difficulties which are present even to-day, it is not astonishing that this apparently simple procedure should have been attended with such a mortality and that its progress should have been slow.

At the end of the last and the beginning of the present century many technical methods were proposed; indeed, almost every conceivable means of connecting

the gastric cavity with the exterior has been suggested. Probably the most striking commentary on the lack of appreciation of the fundamental principles underlying the operation is the fact that so many surgeons, being dissatisfied with the results obtained by the methods in use, have advocated different procedures.

TYPES OF GASTROSTOMY

The first gastrostomies were in the form of a simple cone of stomach brought up to the skin (*Fig. 521*). This method was then modified so that some pressure would be exerted on part of the tube to prevent leakage. Girard¹⁴ and von Hacker¹⁵ brought the cone through the rectus abdominis in the hope that the muscle would act as a sphincter. Hartmann,¹⁷ Frank,¹³ Sabanïeff,³² and, later, d'Agostino⁶ brought the gastric cone in an oblique fashion through the muscles and subcutaneous tissue to the skin, the pressure of the tissues being supposed to cause compression

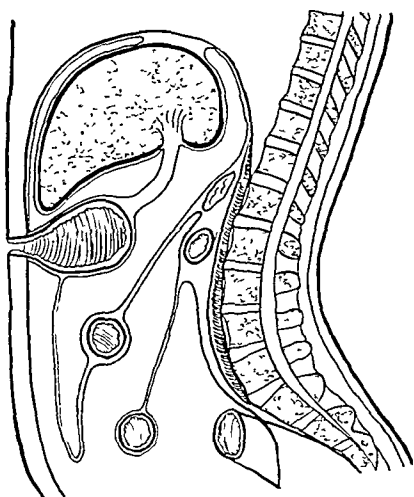


FIG. 521.—Drawing of a sagittal section of the abdomen showing a gastrostomy of the simple cone type. Various forms of gastrostomy—those using the rectus abdominis as a sphincter, those using an oblique path (Frank), and that with the formation of a valve (Senn)—all finally conform to this type.

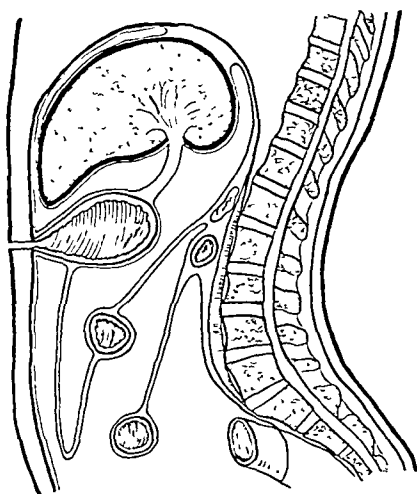


FIG. 522.—Drawing of a sagittal section of the abdomen showing a gastrostomy with a fistulous track through the abdominal wall. All the types of gastrostomy in which there is such a fistula, irrespective of the manipulations of the gastric wall—i.e., simple fistula, Stamm-Kader type, or Witzel-Marwedel form—finally adopt this conformation.

of the cone and so prevent leakage. These methods have no real advantage over the simple operation, since, as pointed out by Depage amongst others, the obliquity of the cone soon disappears, the track finally coming to conform in appearance with the simple cone. Hahn¹⁶ brought the cone between two of the lower ribs. This method has the grave disadvantage that a large amount of the stomach is sacrificed.

A spate of methods now appeared, but for convenience they will be considered apart from their chronological order. A modification of the foregoing is the formation of a 'valve' in the gastric opening. As in the preceding type, surgeons imagined that tissues would remain where they had been placed at operation, and no allowance for alterations in consistency, e.g., following inflammation, was made. Senn³³ suggested a method by which a fold of mucosa or gastric wall could be fashioned at the outlet of the cone. It was thought that such a fold would act like a venous valve. However, the tissues become thickened and fail to act in the desired manner.

Post-mortem examinations, for what they are worth in this respect, show that the folds are actually present, but are quite incompetent. Ullmann³⁸ and Souligoux³⁵ rotated the cone in order to produce a spiral valve effect.

In all the above types the end-result is similar, and although in ideal conditions a good result may be obtained, leakage in the absence of such conditions is always liable to occur, because of the direct continuity of the gastric cavity with the exterior.

Another principle suggested is the connection of the stomach with the exterior by a fistulous track through the abdominal wall (*Fig. 522*). The fistula is kept patent by a tube or some similar apparatus. Such a method, which was one of the first used, has been advocated even recently.⁸

The idea of producing a valve in the stomach in addition to the fistulous track in the abdominal wall has been utilized by Stamm,³⁶ Fontan,¹² and Kader.²³ It

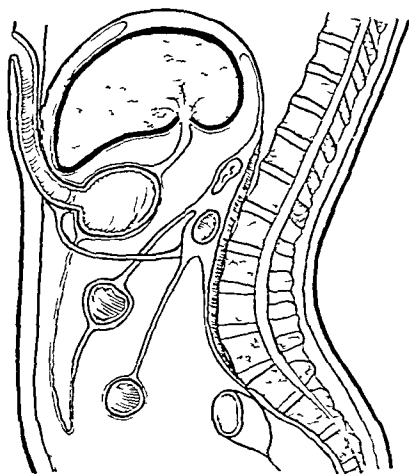


FIG 523—Drawing of a sagittal section of the abdominal cavity showing the relationships of the structures in a gastrostomy formed by a jejunal loop.

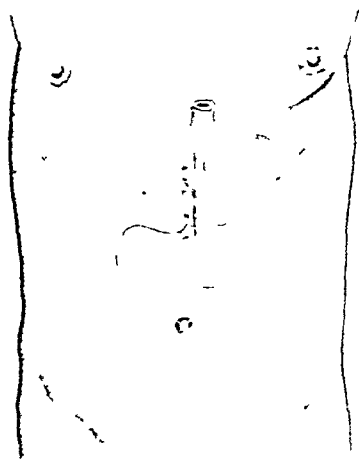


FIG 524—Drawing to show a gastrostomy formed by a jejunal loop (Tavel type).

may be noted in passing that the Stamm gastrostomy is usually attributed to Senn. Kader's gastrostomy is similar to Stamm's and quite unlike Senn's, so that the term Kader-Senn gastrostomy, used so frequently, is incorrect. The failure of the valve action mentioned above in the Senn gastrostomy also applies to the Kader and Stamm forms, etc. Their success is due largely to the tube fitting tightly into the fistula. It is of interest that in the description of some of these operations, although the result is said to depend on a valve action, attention is given specially to the suturing of the wound round the rubber tube.

Another principle, an intramural oblique canal in the stomach wall, was adopted by Witzel⁴¹ and Marwedel²⁵ amongst others. Witzel made the canal outside the muscle, and Marwedel constructed a canal between the muscle and gastric mucosa. Modifications to produce an epithelium-lined canal were also introduced.⁹ In these cases also, partly as the result of the presence of a rubber tube, the canal gradually becomes straightened out so that the end-result differs little from that obtained by the other methods.

All these operations, therefore, notwithstanding the technical differences in their execution, result in a track which is, at least in part, lined by granulation tissue. Such a track readily and rapidly contracts and heals, so that if the tube is left out

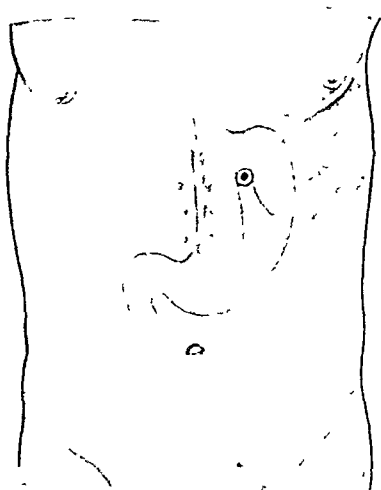


FIG. 525.—Drawing showing the general relationships of stomach, gastrostomy stoma, and wound in cases in which a Janeway operation is performed. The incision need not be in the midline, but investigation of the abdominal cavity is easier through this opening. (After Quirk and Martin.)

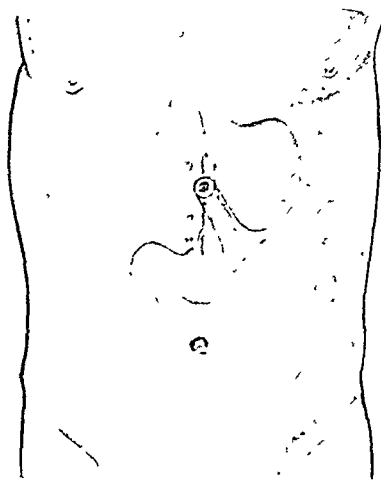


FIG. 526.—Drawing showing relationships in a Janeway gastrostomy when the left hypochondrium is conserved for later procedures. The gastric tube is brought out through the wound instead of through a separate stab incision as in Fig. 525.

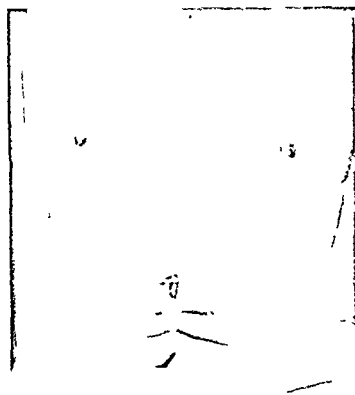


FIG. 527.—Photograph of a Janeway gastrostomy in which the gastric tube was brought through the wound. Cf. Fig. 526.

for only a few hours it may be impossible to re-introduce it. Some kind of apparatus must be worn continually; that such should be made of one of the rarer metals will be no comfort or compensation to the patient. Another disadvantage, only appreciated when further procedures—in the form of an œsophagoplasty for

example—are undertaken, is that the chronic inflammation which occurs in the wall of the fistula causes fibrosis in the surrounding tissue and thus abnormal lines of tension in the region. These changes increase the difficulties of operations in the area, especially if there be a narrow costal margin. The writer has had on more than one occasion to rue the performance of a gastrostomy of this kind.

It was not until the early years of the present century that the principles underlying an ideal gastrostomy were thoroughly appreciated. The essential features are that a narrow track should connect the stomach with skin without encroaching unduly on the stomach or reducing its capacity, and that such track must be lined by epithelium of the intestinal type. This may be achieved in two main ways: (1) Some viscus may be interposed between the stomach and skin; and (2) A tube may be formed from the stomach wall.

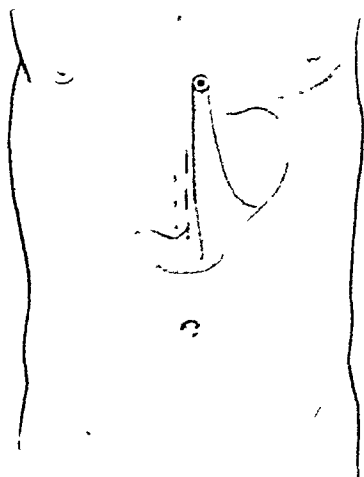


FIG. 528—Drawing to show the relationships of stomach, flap, and incision in the Beck-Jianu gastrostomy. The flap is taken from the greater curvature and the pyloric end of it makes the external opening (stoma). Depending on the amount of greater curvature used, the stoma may be placed just below or above the nipple line. The advantages in cases in which œsophagoplasty is to be performed need no comment.



FIG. 529—Photograph showing the result obtained with the greater curvature flap (Beck-Jianu gastrostomy). Cf. Fig. 528.

A piece of jejunum as the intermediary between skin and stomach (*Figs. 523, 524*) was suggested by Tavel.³⁷ Both jejunum and colon have been used as part of an antethoracic œsophagoplasty, but though these produce ideal gastrostomies, the procedure is greater in magnitude than is justified in most cases, especially in wasted or dehydrated patients.

The use of a tube of stomach wall is probably the best procedure for a gastrostomy. Several modifications have been advocated, a flap being taken in various ways. The Hirsch type,¹⁸ in which a longitudinal flap is taken from the anterior surface of the stomach, does not seem theoretically to be a good one, since the vessels run transversely. Having seen part of the flap slough in one case, the writer has since avoided this method, so that no critical assessment can be offered.

Both the methods in which transverse flaps are used—the Depage,⁷ with the

base of the flap on the lesser curvature; and the Janeway,²⁰ with the base on the greater curvature—are excellent, and indeed the author now uses the second as a routine unless other methods are definitely indicated (*Figs. 525–527*). It is unnecessary to describe them here, as good accounts of the technical details are given in the literature.^{24, 29}

Another method in which the greater curvature is used—the Beck-Jianu gastrostomy^{4, 21}—is excellent where a long gastrostomy tube is required (*Figs. 528, 529*). There are, however, difficulties and possible complications not suggested in the literature.²⁸ That even these forms may leak in certain cases is shown by the fact that modifications of them, e.g., the contriving of valves, etc., are proposed.

Summary.—The various forms of gastrostomy which have been suggested are:—

1. A simple cone of stomach wall.
 2. A cone with (*a*) a sphincter of rectus abdominis, (*b*) an oblique track.
 3. A cone with a valve.
- These all result in a simple cone.

4. A simple fistula through the abdominal wall, with some mechanical device.

5. An abdominal fistula with (*a*) a valve, (*b*) a gastric intramural canal.

These all result in a simple fistula lined by granulation tissue.

6. Some hollow viscus connecting stomach to skin.

7. A gastric flap forming a connecting tube.

The ideal gastrostomy is one which does not leak and which does not require the continual wearing of apparatus. As will be shown, leakage may not be preventable in all cases, but the last two forms will leak much less frequently than the others. The practicability of discarding a rubber tube, except for actual feeding, depends as do other possible desiderata on the lining of the fistula being epithelium, and preferably intestinal, i.e., alimentary-canal, epithelium. Skin often suffers considerably from the action of gastric juice, but the writer has noticed that it gradually becomes accommodated to its environment and appears to be healthy.

About forty different kinds of gastrostomies have been described, so that only a very small proportion of them have been mentioned here. They all fall, however, into one or other of the groups given above.

Author's Cases.—The writer's experience of the operations on which the various observations are based is as follows:—

Simple cone	1
Cone through rectus	3
Frank gastrostomy	3
Souligoux gastrostomy	2
Senn gastrostomy	1
Witzel gastrostomy	3
Marwedel gastrostomy	2
Stamm gastrostomy	10
Tavel gastrostomy (jejunum)	1
Janeway gastrostomy	15
Hirsch gastrostomy	1
Depage gastrostomy	1
Beck-Jianu gastrostomy	7
Total	50

There were four deaths attributable to and occurring within three weeks of the operation. The causes were pneumonia, local peritonitis (Janeway gastrostomy),

acute dilatation of the stomach, and acute œdema of the lungs (Beck-Jianu gastrostomy). The last two were avoidable (*see* later). The age of the patients ranged from 19 to 87 years.

CONSIDERATIONS OF THE PHYSIOLOGY OF THE STOMACH IN RELATIONSHIP TO GASTROSTOMY

In many, if not most, surgical operations one is very inclined to submerge all question of the function of an organ in the morphological achievement. Just as in other situations—e.g., the intestines, the biliary tract, the lungs, etc.—the attention of surgeons to the study of physiology is compelled here also. Although many observations on the stomach have been made, the lack of understanding of gastric phenomena is soon appreciated by any surgeon who has had to cope, or attempt to cope, with the cases of uncontrollable leakage which arise in any series of gastrostomies.

Both local conditions—motility, secretion, and sensation—and general conditions, especially the relationship of gastric activity and hæmatopoiesis, require and repay study.

Gastric Motility.—Ordinarily food is passed from the cardia to the pyloric antrum by the 'tonic' contraction of the muscular coats of the stomach. The only obvious contractions are 'ripples' which may be discerned in the wall. This tone of gastric muscle allows accommodation of the organ to even relatively sudden augmentation of the contents, and thus usually intragastric tension is not high. When there is a spasm of part of the stomach the pressure rises, and this phenomenon will be discussed later. That intragastric tension, even during digestion and the passage of food into the duodenum, is not high is shown by the fact that no matter what form of gastrostomy is employed, in a proportion of cases no leak will occur. Indeed, the success of a gastrostomy, in a general way, is due not to niceties of intricate technique but to this fact. On the other hand, with those abnormal cases which will be discussed, whatever the form of gastrostomy, leakage is inevitable in our present state of knowledge.

It goes without saying that any present form of feeding by gastrostomy is not as advantageous as the normal method, since the food is not delivered directly into the cardiac region. However, this does not seem to matter as much as one would expect; the stomach is able to accommodate in an astonishing manner. I have not been able to detect any superiority of a gastric opening placed as high as possible in the stomach over those in which (to allow room for a subsequent œsophagoplasty) the gastrostomy was placed near the pyloric antrum.

THE GASTRIC PATHWAY.—That material passes along the lesser curvature of the stomach by way of a '*Magenstrasse*' was emphasized by Aschoff.¹ This 'pathway' is well marked in some animals, though there have been differences of opinion as to its importance in human beings. Two personal observations are most readily explicable on the hypothesis of such a pathway (or some modification of it).

Case 1.—A female aged 67 years, suffering from carcinoma of the œsophagus, had had a Janeway gastrostomy performed. About a week after the operation the food passed into the tube began to leak back. Two days later the whole of the food was returning. She had had complete œsophageal obstruction, but it was thought that with the rest to the œsophagus it might be possible for her now to swallow. A feed of 12 oz. was given through the gastrostomy and almost immediately leaked back. She was then given 12 oz. of similar (liquid)

food by mouth, which she was able to swallow, and it did not escape either then or later. After two days she was able to take food again through the gastrostomy tube.

Case 2.—A male aged 54 years, with a carcinoma of the œsophagus, had had a Stamm gastrostomy performed. At times he complained of fullness after meals. On two separate occasions, having been given 12 oz. of a distinctive food (liquid), he felt uncomfortable and vomited. The vomitus, several ounces in amount, was quite unlike the food given through the gastrostomy. Incidentally, the œsophageal stricture was not a complete one and there was no evidence whatever of accumulation of material above the growth.

These cases suggest that food passed through the gastrostomy tube ran into a loculated area of stomach. Of course the limits of this area might have been determined by a transverse rather than a longitudinal contraction of the stomach wall (as postulated to account for the gastric pathway), but *Case 1* is difficult to explain on such a hypothesis. In addition X-ray examinations showed that the gastrostomy tube passed upwards towards the cardia and a barium meal passed apparently uninterrupted along the length of the stomach.

Since the majority of gastrostomies are made on or near the greater curvature, the integrity of the gastric pathway may help to account for the absence of leakage during ordinary digestion. In the cases examined by the writer, a barium meal passed first up to the cardiac end of the stomach, though it has not been possible as yet to determine the subsequent course of the meal in relationship to the curvatures.

There are two mechanical factors which may play a part in determining the continence of a stoma. The first is the pliability of the gastric wall and the possibility of a neighbouring area becoming opposed to the opening. The other is the extraordinary elasticity of the mucous membrane, so that folds of mucosa arising at a distance from the stoma may act as valves. Since, however, as will be shown, if other factors are not favourable leakage will occur, these factors cannot be of any great import. In any case they are dependent on the degree of distension of the stomach.

In certain cases, as mentioned, gross leakage does occur. There is no doubt that the primary cause of leakage is always an increase in intragastric tension. During the act of vomiting there may be no escape of material through the gastrostomy if the œsophagus be patent. If, however, there is a complete obstruction, e.g., from cicatricial or carcinomatous stricture or after œsophagectomy, vomiting is associated with propulsion of material out of the opening. In other cases where there is a spasm of the pylorus and pyloric antrum and even of the main part of the stomach, pressure on the opening, or, where there is a gastric tube as in the Beck-Jianu type, pressure on this tube, will fail or be but temporarily successful in preventing escape of gastric contents. When no food is present even gastric juice will escape.

The nature of the food placed in the stomach plays a role the actual import of which at present cannot be adequately gauged. A number of patients have stated that they have felt better immediately after their first meal of solid food (given by a grease-gun). They felt more comfortable in the epigastrium, they felt "more satisfied", and within a day or two were able to do more physical exercise. One patient who had been in the habit of feeding himself with liquid food for two years, asked when leaving hospital, after having had solid food for a week, if he could take a grease-gun with him, because he felt so much better. It is not necessary here to discuss the mechanisms (stimulation of peristalsis, stimulation of secretion, etc.) by which this effect is produced.

Another important factor in the mechanics of intestinal activity is the presence of gas in the bowel. One is at first astonished to see the amount of air, mixed with saliva, which escapes from an œsophageal opening (e.g., after œsophagectomy). Although no proof can be offered at the moment, I feel convinced that one of the reasons why patients receiving solid food feel much less discomfort than those having liquid food is that air contained in the interstices of minced food imparts an elasticity which is absent from liquid. Incidentally, patients having liquid food feel better when mildly effervescing mixtures are given after their meal.

Lastly, the association of secretory activity with the motility of the stomach must always be considered:—

A female, aged 44 years, had had a gastrostomy performed for dysphagia from carcinoma of the œsophagus. Ten days later she was still only able to take less than 6 oz. of her food at each meal. The full amount (12 oz.) contained 2500 calories per day. Various methods of treatment were tried without success, but when she was given hydrochloric acid with her food (she was found to have achlorhydria) she was able immediately to take the full amount—12 oz.—without discomfort or pain.

A man aged 54 years, who had had an œsophagectomy performed for carcinoma, developed a leak from his gastrostomy. His stomach was found to be extraordinarily sensitive to chemical stimuli. Immediately after the passage of a small amount (1–2 oz.) of sodium bicarbonate solution into the stomach, two or three ounces of bile-stained fluid could be withdrawn. If hydrochloric acid was run in, no bile could be aspirated. Incidentally, leakage from the gastrostomy was most marked when too much acid was given with the food. This also occurred when no acid was given, and for some weeks he presented a difficult mathematical problem at meal times.

Secretory Activity.—Disturbances of secretory activity of the stomach are inevitable in most cases. Not only is there some lesion of the œsophagus, e.g., an ulcerative condition which predisposes to infection of the gastric mucosa, but the actual diminution in the intake of food or the swallowing of food in an unusual state (as when obstruction becomes marked) must have its effect on the stomach. In addition, the presence of a foreign body—the rubber tube, even if not continually worn—produces some degree of gastritis. Some inflammation, observed either macro- or microscopically, has been present in all cases so examined.

Which factors are principally responsible it is not possible to say, but all cases specially investigated showed a marked diminution of gastric acidity, and in most of them there was an achlorhydria.

How this may affect motility has been mentioned already.

Sensation in the Stomach.—It is not proposed to debate the vexed question of gastric sensation. As far as can be determined, sensations observed by patients with gastrostomy are strictly comparable with those occurring in normal individuals, though when present they are usually felt in a heightened degree. This last is probably because the stimulus is greater than in a normal person since food passes into the organ more rapidly. It is of course important that material placed in the stomach should be administered slowly. It is possibly noteworthy that sensations of discomfort and epigastric pain, especially on overfilling the stomach, in two patients who had had both vagi cut during œsophagectomy, were the same as experienced previously.

Relationship of Stomach to the Lower Alimentary Canal.—The most obvious disturbance, in this respect, is ‘gastric’ diarrhœa, which is not uncommon in these patients. Administration of acid not only cures these cases but also

improves many that have mild lower abdominal colic, etc., without actual diarrhœa. Although the problem has not been investigated at all thoroughly, it has been found that when a gastrostomy is functioning well intestinal disturbances are at a minimum, and that these are most marked when some point in the management of the gastric fistula has been overlooked.

Relationship of Gastric Activity to that of Other Organs.—As has been shown in recent years, the most important general relationship is that of stomach with bone-marrow function. Its importance is reflected in the anæmia which is present in the majority of the patients who require gastrostomy. Associated with this is a low blood-pressure. Although the matter is not susceptible, at present, of scientific proof, I feel satisfied that anæmic patients subjected to gastrostomy (e.g., those with œsophageal carcinoma and associated achlorhydria) recover from anæmia more quickly when given stomach extracts (intrinsic factor) in addition to other treatment.

The anæmia and its treatment is especially significant when some major procedure, e.g., œsophagectomy, is contemplated. The oxygen and carbon dioxide carrying capacity of the blood is of paramount importance during an operation where vital capacity is reduced, and other factors, such as severe shock, weigh greatly against a patient who has but little reserve. The question of low blood-pressure is bound up in some measure also with the problem of the blood-proteins, and thus with diet.

INDICATIONS FOR GASTROSTOMY

The indications for gastrostomy fall into two main groups: (1) Where such a procedure is an integral part of the treatment, e.g., prior to œsophagectomy or to enable retrograde bouginage of an œsophageal stricture, etc.; (2) As a palliative measure. It is unnecessary to discuss the first group. In the second, if complete œsophageal obstruction be present, the urgency of the problem overshadows other considerations. In less severe dysphagia, especially if due to carcinoma, it is often said that the patient's last few months are made more miserable by a gastrostomy. Reasons for this are leakage, necessity for wearing a tube, and the abnormal method of feeding.

The gastrostomy does not usually leak if it is managed correctly—i.e., if food is given slowly, if the proper kind is given, and if the degree of acidity is controlled, etc. The necessity for wearing a tube depends on the type of gastrostomy. The method of feeding is not really objectionable if the patient feels hungry and is interested in his meals; and this is so especially when solid food is given. Another point, often overlooked, is the fate of the gastrostomy opening when (as frequently happens) the patient recovers his ability to swallow. In these circumstances the patient may remove his tube and swallow his food until dysphagia again supervenes. By this time the gastrostomy has closed, and, as happened in one case in this series, it is necessary to make another.

The real objection to the operation is therefore bound up in the form of gastrostomy adopted. With a gastric-flap gastrostomy there is no need for the presence of a tube all the time; and even if this be left out for some weeks or months and the opening contracts, it is a very simple matter to dilate this sufficiently to allow the easy introduction of the tube once more.

The great improvement in the physical and mental condition of the patient after adequate gastrostomy feeding is sufficient justification and compensation for the operation. The indication for gastrostomy, therefore, with a few exceptions to be discussed, is intractable dysphagia of whatever degree, if due to organic disease of the œsophagus. It is not necessary to enumerate these conditions or to give a list of other diseases which may affect the œsophagus secondarily.

CONTRA-INDICATIONS TO GASTROSTOMY

This is a difficult problem, and it is doubtful if it is practicable at present to give an adequate account of the cases in which gastrostomy should not be performed.

It is extremely mortifying, and indeed heartbreaking, to attempt to deal with the fortunately few cases in which, despite a satisfactory gastrostomy and treatment,



FIG 530—Photomicrograph of a section of a carcinoma of the lower part of the œsophagus which had invaded the vagi and splanchnic nerves. In this case there was such a condition of the stomach that, despite all treatment, uncontrollable leakage occurred. (The gastrostomy was of the Beck-Jianu type.) O, Esophageal wall, the tumour invading the muscle coat, P, A subpleural plaque of carcinomatous tissue, L, Lung adherent to the growth, N, A large nerve (vagus) surrounded by growth—there is well-marked perineural fibrosis (Hæmatoxylin and van Gieson stain) ($\times 28$)

gross leakage occurs. It is quite impossible to control the flow of food and gastric juice from the opening. Such cases have been referred to by other writers.^{2, 42} Such a complication, which constitutes the major contra-indication to the operation, occurs most frequently with extensive carcinomata of the lower end of the œsophagus and of the cardia of the stomach. It is suggested that irritation of nerves by the tumour results in spasm of some part of the stomach. Certainly post-mortem examinations in such cases have revealed a gross involvement of the vagus and splanchnic nerves (*Fig. 530*). I hesitate to perform a gastrostomy in a patient with a carcinoma low in the œsophagus if some symptom, usually pain, is present indicating involvement of surrounding tissues by growth.

Barber² has observed a spasm of the pyloric antrum at operation in such cases. If the condition be recognized and dysphagia is marked, then jejunostomy should be performed in preference to gastrostomy. This condition, especially as a contra-indication to gastrostomy, is not sufficiently recognized.

Another contra-indication is an advanced state of malnutrition in a weak patient, especially if there are pulmonary or circulatory complications. It is astonishing how an apparently moribund individual may be restored after gastrostomy to a relatively normal condition; but this applies mainly to examples of acute complete obstruction. On two occasions, having arranged for patients to be admitted to hospital, a delay of three or four days occurred. As the result of dehydration, complete obstruction having supervened, the patients when they arrived were scarcely recognizable as the same people. Following gastrostomy they recovered rapidly. In the more chronic forms, which have been neglected, the prognosis is more grave. Infection of the wound or the peritoneum is liable to develop, and in any case cardiac failure or pneumonia is not far distant and may be precipitated by the operation. The records of almost any hospital will reveal post-operative fatalities in this type of case. If the general condition cannot be improved by the parenteral administration of fluid and salt in sufficient amount, then gastrostomy is inadvisable.

DIET IN GASTROSTOMY

A few moments' contemplation compels the recognition of the formidable responsibility placed upon the surgeon in these cases; namely, the taking over of one of nature's most complex phenomena—the choice and the determination of the quantity of food and drink. This everyday marvel is passed over by most of us probably because it is so commonplace. The salting of food 'to taste', the migration of animals to salt country, hunger for a special kind of food, e.g., fat, if it has been absent from a diet, and the cannibalistic habits of fish in inland salt seas or lakes (not for the flesh, but for water with a relatively low salt content) are some of the many examples which could be quoted of this kind of 'automatic' eclecticism. In the case of gastrostomy feeding, what the patient ordinarily is able to do unconsciously must be painstakingly controlled by the surgeon.

Liquid Diets.—The first question is that of the amount of food. This must be calculated carefully on the basal metabolic requirements of the individual, with any additional amount that may be necessary in order to ensure that the patient will regain his weight. The writer's practice is to calculate that amount on the patient's original, rather than his present, weight. That the amount should be calculated accurately is a point that requires emphasis. Any feeding that is performed by guess or rough estimate will be found almost invariably on investigation to fall short of the necessary quantity, sometimes astonishingly so. On one occasion, having been asked to give an opinion on a case of carcinoma of the œsophagus, I found on calculation that the amount of food being given corresponded to 500 calories; actually he should have been having 2000. Fortunately such incredible discrepancies are not common; but investigation will show some discrepancy in all uncontrolled diets.

The proportion of protein, carbohydrate, and fat should be considered in the preparation of meals. The difficulty with high caloric fluid diets is one of providing

sufficient protein. In addition to the lack of total amount some of the amino-acid groups are present only in small amounts in the material available in fluid food, and if such only are given, adjuvants are necessary to make good the deficiency. A great deal of time and effort has been expended in attempts to find the most satisfactory means of supplying these, but as a simpler method (the use of solid food) has been used and found sufficient, no discussion of them is necessary.

Vitamins are not likely to be overlooked in these times, but the writer has seen one case of incipient scurvy, and has had two cases of secondary pellagra in his own practice. That cases of œsophageal and gastric disease, especially carcinoma, are prone to develop pellagra has been recorded in the literature.³⁴

The amount of salts, sodium chloride especially, must always be given attention. Iron is necessary when anæmia is present, and if this be administered in a pure form, copper, manganese, etc., require consideration.

Actually when fluid feeds are given there are so many factors requiring attention, and the necessity for and absence of many of them are so difficult of diagnosis or estimation (especially in the early stages), that a few patients would keep occupied a special dietetic department, a biochemist and several nurses, to say nothing of the harassed surgeon, for most of the day. And yet all these things and probably others are of great importance, and their omission (and even the absence of any one) conduces to the typical 'poor condition' of the gastrostomy patient.

Solid Diets.—An immense difference is made, however, if solid food is supplied. The giving of such food by means of a grease-gun has been suggested by a number of writers.^{27, 28, 30} But it has been advocated as merely an alternative to liquid feeds because of the convenience to a patient out of hospital or for economic reasons. Certainly it is easier to prepare, is much cheaper, is easily given, and the articles required are those in everyday use in an ordinary household. These reasons, however, important though they are in themselves, are not the material and commanding grounds for its adoption.

The solid feeds, which of course must also be calculated carefully, contain a larger amount and a greater variety of protein; vitamins are present in the fresh food, and so have not to be added specially; adequate amounts of salts, e.g., those of iron and magnesium, are contained in the food. The importance of the physical and chemical state of substances, especially proteins, in the fresh and solid state in relationship to digestion, absorption, and metabolism is not understood; that there are differences between solid and liquid foods is tacitly admitted by any hungry person who chooses a beefsteak rather than an egg-flip.

Patients (from the psychological point of view) feel better immediately, and from the beginning of their receiving solid food take a greater interest in their treatment. This has been an almost constant observation in the patients so treated. They become brighter and more hopeful in their outlook. This is of extreme importance when further operative procedures are intended. The improvement in the local condition is also striking. The patient feels more comfortable, is able to take a larger amount of food (as estimated in calories), and is much less liable to epigastric pain or discomfort. The 'comfortable' or satisfied feeling experienced seems to be distinct and differentiable from the absence of pain; it is a positive sensation of well-being, and is the feature most often referred to by the patient—indeed it is frequently remarked upon, being one of the subjects of daily conversation.

There is also an improvement in the general physical condition. The most obvious and easily demonstrable features are the rise in blood-pressure (when this has been abnormally low) and the improvement in the blood-cell count and the hæmoglobin content of the blood. The improvement is also reflected in the patient's ability to perform mild exercise, and, more important, to withstand operative interference more easily and satisfactorily. The onset of signs of shock—falling blood-pressure and rising pulse-rate—is always later at operation in these patients than in those who have had fluid food only.

Two statements of Beaumont are worthy of note here: "Bulk, as well as nutriment, is necessary to the articles of diet;" and "solid food, of a certain texture, is easier of digestion than fluid".

Importance of Saliva.—That the introduction of saliva into the stomach in cases of complete œsophageal obstruction is important is a conclusion that has been reached by a number of observers.^{5,19} It is mentioned here to emphasize its importance. Incidentally it demonstrates one point that is frequently overlooked. Lack of attention to any one matter nullifies all the other efforts that have been made to deal adequately with the problem. Failure to achieve a good result in such circumstances leads to the opinion that these factors are not important; this of course is not so. All factors must be dealt with to obtain a good result.

Incompatibilities of Diet.—Every now and again one encounters a more or less severe upset in a patient who has had every care and in whom there is no obvious cause for the departure from normal. This consists of abdominal pain or discomfort, regurgitation of food from the stoma, diarrhœa, etc.

The problem is a difficult one, and may be quite disturbing until some article of diet is found responsible, when its exclusion obviates the trouble. Some little time usually elapses before the culprit is recognized; the diet and adjuvants should be checked carefully, the acid content of the stomach juice re-investigated, etc. It is always advisable to discover, if possible from the patient, whether he has ever reacted unusually to any particular kind of food. Care must be taken not to suggest any particular substance as the cause of the disturbance; in one case the patient became ill whenever she knew that one particular form of egg-flip was given, but there was no trouble when this was disguised. True incompatibilities have been due, in different cases, to tomato, orange juice, white of egg, etc.

If fluid feeds are given, the requisite number of calories are made up by disproportionately large amounts of fat and carbohydrate. In three cases, with cholelithiasis, the high fat feeding was followed quickly by attacks of biliary colic, and in one case jaundice.

THE OPERATION

The actual operation and the associated immediate treatment, though intrinsically important, is considered last because, besides the other features, it fades relatively into insignificance.

Pre-operative Treatment.—This consists largely in administration of fluid rectally, in more dehydrated cases subpectorally, and in the worst cases even intravenously. It is to be noted that greater care must be exercised in the more grossly dehydrated patients not to restore fluid too rapidly, otherwise œdema, especially œdema of the lungs, will supervene.

Anæsthesia.—Undoubtedly the best anæsthesia is some form of local administration. The writer's practice is, after giving paraldehyde rectally, to produce a block anæsthesia of the lower thoracic nerves at the rib margins with a $\frac{1}{2}$ per cent solution of novocain (or some similar substance), and to follow by the introduction of a 1 per cent solution into the peritoneum round the stomach. The upper part of the abdomen should always be investigated for complicating conditions, e.g., metastases, at the time of operation. The anæsthesia produced is adequate both for this and for the actual procedure on the stomach.

Post-operative Treatment.—This consists of the administration of an adequate amount of fluid in the same way as that given before operation. Feeding may be commenced immediately after the return of the patient to the ward. Three ounces is given three-hourly for the first day, and this is increased by an ounce a day until the patient is taking full feeds. Other treatment, such as is routine for all operations, is also given.

Complications.—*Post-operative shock* varies almost directly with the general condition of the patient. This may be overcome by using the form of gastrostomy which demands, within the requirements for a good result, the smallest amount of manipulation, by gentleness of handling, and rapidity of operating. A Depage or Janeway gastrostomy may be completed in comfort in less than half an hour, and in the great majority of cases the patient has the same pulse-rate and blood-pressure at the completion as at the beginning of the operation.

Pneumonia or some degree of pulmonary collapse is not uncommon after gastrostomy, though occurring almost entirely in the more poorly nourished individuals. One death occurred in the writer's series from this cause. Post-operative administration of carbogen should be routine treatment.

Wound infection depends almost entirely on the general condition of the patient. It is desirable to use a small drain tube in the wound of those patients who are liable, as can usually be foretold, to develop such a complication.

Peritonitis, like wound infection, depends on the condition of the individual. The one fatal case from this cause occurred in a patient who, in retrospect, should not have been operated on. Incidentally, in no case was there any indication of leakage of a suture line even in cases where this was long (Beck-Jianu type) or where acute dilatation of the stomach had occurred.

Acute dilatation of the stomach occurred four times. The essential point in its treatment is to keep the stomach empty (an easy matter when there is a gastric fistula), together with parenteral administration of adequate amounts of fluid. It is necessary, however, that the diagnosis should be made early. Any regurgitation of dark material from the stoma should immediately be followed by aspiration of the stomach contents. Also any sudden increase in pulse-rate should lead to an immediate investigation of the stomach. It is astonishing that a stomach may contain one or two quarts of fluid without there being any leak from the stoma. Gastric dilatation usually develops within a few hours of the operation, but I have observed it as late as the tenth day. If the stomach is kept empty and fluid given by other routes, most if not all cases will recover. The one death from this cause took place because a sudden increase in pulse-rate from 80 to 120, occurring at two o'clock in the morning two days after operation, was not notified, and when seen at 8 o'clock the patient had a pulse of 140. At this time treatment was of no avail.

Complications due to some method of treatment may arise, and of these that due to the too rapid administration of fluid is the most important. A rising pulse-rate not explicable by some obvious cause should suggest the discontinuance of fluid. In three cases this was followed by a return of the pulse-rate to normal; fluid was then given again more slowly without ill effect. The one death from this cause (from acute œdema of the lungs) occurred after the pulse-rate had been rising gradually for several hours without its significance being recognized.

Leakage from the stoma, and cicatrization, stenosis, and closure of the wound, have already been discussed.

SUMMARY

The operation of gastrostomy is one that requires special knowledge, care, and study, not because of any difficulties in the procedure itself, but on account of the innumerable problems to be faced after the immediate operative result has been attained.

No gastrostomy can be regarded as justifiable unless the result striven for is satisfactorily achieved, i.e., that the patient receives adequate and satisfying nourishment and obtains this without discomfort and distress. The indications for the operation are bound up with these requirements.

The particular operative method is of relatively little moment in ideal circumstances, though certain forms, especially those which utilize a gastric flap, are generally more advantageous than others. The control of the stoma, however, depends ultimately on two factors: (1) The stomach is not an inactive receptacle for food, but is a physiologically active organ—hence the satisfactory functioning of a gastrostomy depends more on a 'normal' state of gastric tone and secretion than on the nature of the stoma. Any departures from normal must be rectified or compensated for. (2) Diet must be carefully and thoroughly controlled, and is best given in the form of solid food.

A case of gastrostomy therefore demands minute attention—greater than most major operations—if the many requirements are to be fulfilled. It is only in these circumstances that the patient will gain that amount of comfort and improvement in his physical and mental condition which justifies the operation as a palliative measure, that the characteristic 'poor condition' will be obviated, and that the maximum foundation for withstanding any other procedure will be ensured.

REFERENCES

- ¹ ASCHOFF, L., *Lectures on Pathology*, 1924. New York: Paul B. Hoeber.
- ² BARBER, H. W., *Ann. of Surg.*, 1935, cii, 375.
- ³ BEAUMONT, W., *Experiments and Observations on the Gastric Juice, and the Physiology of Digestion*, 1833. Plattsburgh: F. P. Allen.
- ⁴ BECK, C., and CARRELL, A., *Illinois Med. Jour.*, 1905, vii, 463.
- ⁵ CARLSON, A. J., *Amer. Jour. Physiol.*, 1915, xxxvii, April, 50.
- ⁶ D'AGOSTINO, F., *Arch. ital. di Chir.*, 1921, iii, 285.
- ⁷ DEPAGE, A., *Cong. franç. de Chir.*, 1903, xvi, 332.
- ⁸ DRAGSTEDT, L. R., HAYMOND, H. E., and ELLIS, J. C., *Surg. Gynecol. and Obst.*, 1933, lvi, April, 799.
- ⁹ EDWARDS, *Trans. Illinois Med. Soc.*, 1894, xlv, 131, quoted by Quick and Martin (loc. cit.).
- ¹⁰ EGBERT, in *Petit's Traité de Gastrostomie*. Paris: V. Adrian Delahaye et Cie., 1879.
- ¹¹ FENGER, E., *Arch. f. pathol. Anat.*, 1854, vi, 350.

- ¹² FONTAN, J., *Dixième Cong. franç. de Chir.*, 1895. Paris.
- ¹³ FRANK, R., *Wien. klin. Woch.*, 1893, vi, 231.
- ¹⁴ GIRARD, C., *Cor.-Bl. f. schweiz. Aerzte*, 1888, xi, 345.
- ¹⁵ HACKER, V. R. VON, *Wien. klin. Woch.*, 1890, iii, 827.
- ¹⁶ HAHN, E., *Zentralb. f. Chir.*, 1890, xvii, 827.
- ¹⁷ HARTMANN, H., *Bull. et Mém. Soc. de Chir. de Paris*, 1897, xxxiii, 253.
- ¹⁸ HIRSCH, M., *Zentralb. f. Chir.*, 1911, xxxviii, 1561.
- ¹⁹ JACKSON, C., *Arch. of Pediat.*, 1923, xl, 324.
- ²⁰ JANEWAY, H. H., *Jour. Amer. Med. Assoc.*, 1913, lxvi, 93.
- ²¹ JIANU, A., *Deut. Zeits. f. Chir.*, 1912, cxviii, 383.
- ²² JONES, S., *Lancet*, 1875, i, 678.
- ²³ KADER, B., *Zentralb. f. Chir.*, 1896, xxiii, 665.
- ²⁴ MARTIN, H., and WATSON, W. L., *Surg. Gynecol. and Obst.*, 1933, lvi, 72.
- ²⁵ MARWEDEL, G., *Beitr. z. klin. Chir.*, 1896, xvii, 56.
- ²⁶ MAURY, F. F., *Amer. Jour. Med. Sci.*, 1870, lix, 365.
- ²⁷ MAYLARD, A. E., *Lancet*, 1908, i, 1407.
- ²⁸ PACK, G. T., *Surg. Gynecol. and Obst.*, 1933, lvii, 81.
- ²⁹ QUICK, D., and MARTIN, H. E., *Ibid.*, 1928, xlv, 426.
- ³⁰ RUTHERFORD, R., *Brit. Med. Jour.*, 1935, i, 1221.
- ³¹ SÉDILLOT, C., *Gaz. méd. de Paris*, 1847, 90.
- ³² SABANĬEYEFF, I. F., *Chir. Vestnik*, 1893, iii, 690.
- ³³ SENN, E. J., *Jour. Amer. Med. Assoc.*, 1896, xxvii, 1142.
- ³⁴ SIMPSON, S. L., *Quart. Jour. Med.*, 1935, iv, No. 14, 191.
- ³⁵ SOULIGOUX, C., and BLOCH, R., *Presse méd.*, 1920, xxviii, 857.
- ³⁶ STAMM, M., *Surg. Gynecol. and Obst.*, 1914, xviii, 527.
- ³⁷ TAVEL, E., *Arch. prov. de Chir.*, 1906, xv, 317.
- ³⁸ ULLMANN, E., *Wien. med. Woch.*, 1894, xlv, 1662.
- ³⁹ VERNEUIL, H., *Bull. de l'Acad. de Méd.*, 1876, 1025.
- ⁴⁰ WATSON, J., *Amer. Jour. Med. Sci.*, 1844, viii, 309.
- ⁴¹ WITZEL, O., *Zentralb. f. Chir.*, 1891, xviii, 601.
- ⁴² WOLFER, J. A., *Ann. of Surg.*, 1935, ci, 718.

EXPERIMENTAL SURGERY

EXPERIMENTAL LESIONS OF THE RABBIT'S APPENDIX *

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THE experiments described in this paper were done in an attempt to determine the ætiology of acute appendicitis. Muir¹ states that "although appendicitis is essentially a bacterial infection, there is still want of knowledge with regard to the particular conditions which lead to its occurrence".

It is not possible to discuss in detail the extensive literature on this subject. This has already been done by Aschoff.² Some workers, e.g., Adrian,³ Poynton and Paine,⁴ Rosenow,^{5, 6} suggest that there is a specific blood-borne infection; others, e.g. Hilgermann and Pohl,⁷ Weinberg *et al.*,⁸ suggest a specific enterogenous infection. There are, however, many workers, e.g. Aschoff,² Heile,⁹ Williams and Boggon,¹⁰ who are opposed to the theory of a specific infection and stress the mechanical stagnation of the appendicular contents and consequent infection with bacteria normally present in the appendix.

EXPERIMENTAL

I have used for these experiments young rabbits weighing between 500 and 900 grammes. The rabbit is the only easily available laboratory animal which has an appendix at all comparable with that of man. Appendicitis occurring spontaneously in rabbits has been described by Mori.¹¹ The adult rabbit has a large appendix, 8 or 9 in. long, but in the young rabbit it is relatively smaller and thicker walled, being about 3 in. long in the size of rabbit used in these experiments. It is similar to the human appendix in appearance and microscopical structure, with the exception that there is a meso-appendix throughout its length joining it to the jejunum. The blood-vessels in the meso-appendix are quite unlike those of the human meso-appendix in that there is an extensive anastomosis between them and the jejunal vessels. *Fig. 531* makes clear this point—the arterial system is shown injected, and branches of the appendicular vessels can be seen joining directly branches of the jejunal vessels. The double blood-supply is of importance in the study of gangrenous appendicitis.

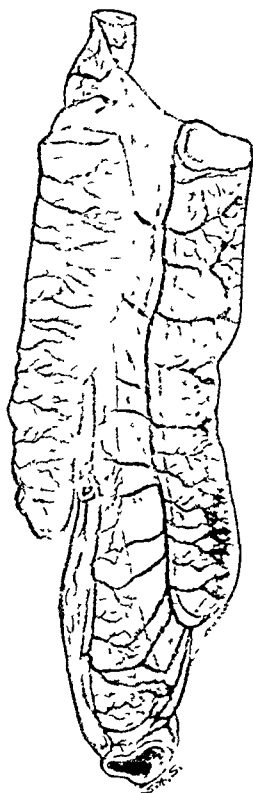


FIG. 531.—The appendix and part of the rectum and small intestine, the arterial system of which has been injected with hydrokollag. The free anastomosis between branches of the appendicular and jejunal vessels is shown.

* This work was done during the tenure of the Cattlin Research Fellowship.

The normal appendix of the rabbit may or may not contain fæcal material; if present, the fæces are semi-solid. In no instance was a fæcolith found.

The bacterial flora does not materially differ, either in quantity or quality, from that of the cæcum. Aschoff² has described a difference between the bacteria present in the proximal and distal parts of the human appendix. No such difference was found in the rabbit.

Experiments Involving Injection of Bacteria.—In the first series of experiments, bacteria were injected either intravenously or into the lumen of the appendix. The bacteria were streptococci (non-hæmolytic) isolated from inflamed human appendices and gall-bladders; hæmolytic streptococci from the throat of patients with tonsillitis; anaerobic bacilli from inflamed human appendices; and the organisms in an emulsion of the purulent contents of human appendices immediately after their removal by operation. The number of bacteria injected varied from 5 to 5000 million. Some of these animals died, but in no instance was there any inflammation of the appendix even when the bacteria had been injected directly therein.

Experiments Involving Damage to the Mucous Membrane of the Appendix.—The appendix was crushed in five rabbits. The lesion was made at about the mid-point of the appendix, and was sufficient to sever the mucous membrane and to produce some

FIG. 532.—Gangrenous appendicitis. Death occurred two days after ligation of the appendicular blood-vessels and meso-appendix.

hæmorrhage in the submucosa. None of these animals showed any sign of appendicitis. At post-mortem, about one month later, the appendix showed a small line of fibrosis at the point where the crushing was made, but there was no inflammation.

Experiments Involving Damage to the Mucous Membrane of the Appendix and Injection of Bacteria.—In another five rabbits the appendix was crushed in a similar way to those in the previous series, and at the end of the operation bacteria were injected either intravenously or into the appendix. The bacteria injected were similar to those used in the first series. None of these animals developed appendicitis.

Experiments Involving Interference with the Blood-supply.—The appendicular artery and vein were ligatured in three rabbits. In no case was there any obvious change in the appendix one month later. A similar result followed from ligation of the meso-appendix.

In four rabbits the appendicular vessels and the meso-appendix were ligatured. Gangrenous appendicitis followed, and death occurred within three days. Fig. 532 shows a gangrenous appendix from one of these rabbits.

Experiments Involving Kinking of the Appendix.—In four rabbits the appendix was kinked on itself, the proximal and distal parts being sutured together with catgut. These animals were killed after one month. At autopsy there was no inflammation of the appendix, which had remained kinked.

Occlusion of the Lumen of the Appendix by Ligature.—The effect of tying a ligature round the root of the appendix depends on whether or not the appendicular blood-vessels are included in the ligature. Fourteen rabbits were operated on in this series, the ligature including the vessels in four, and leaving



the vessels untied in ten. The four whose vessels were tied showed no signs of illness and were killed between one and two months after operation. The appendix showed no signs of inflammation; it was pale and atrophied, as illustrated in *Fig. 533*.

The ten whose vessels were left untied showed a different picture. At about the end of the second week in every animal a lump was felt in the abdomen. This increased slowly in size, until in one case it became the size of a lemon. Eight of the ten animals did not appear ill. They fed normally and increased in weight. They were killed at



FIG. 533—Atrophy of the appendix, thirty-one days after ligation of the root of the appendix and the appendicular blood-vessels.

intervals ranging from two weeks to two months after operation. (The other two died three days after operation with an acute appendicitis and peritonitis. These two will be considered in the next series.) At post mortem the appendix was much enlarged, the degree of enlargement varying with the length of time elapsed since operation. *Figs. 534* and *535* show two of these enlarged appendices, one a month and the other two months after operation. The wall was thin and in some cases loculated. The contents of the appendix consisted chiefly of mucus, together with a small amount of fæces and fæcal bacteria, and a few leucocytes. This mucocele is similar in appearance to a mucocele of the human appendix.



FIG. 534—Mucocele of the appendix, sixty-four days after ligation of the root of the appendix. The ligation did not include the appendicular blood-vessels.

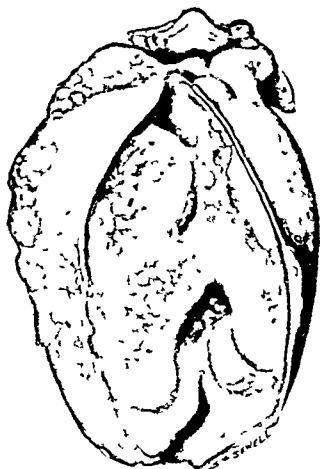


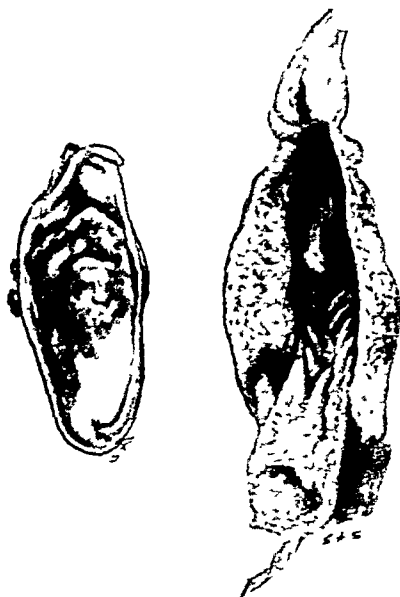
FIG. 535—Mucocele of the appendix, twenty-eight days after ligation of the root of the appendix.

Occlusion of the Lumen and Damage to the Mucous Membrane of the Appendix.—Fourteen animals were operated on in this series. The root of the appendix was ligated as in the last series, the ligation either including the

vessels or not, and the mucous membrane was damaged by crushing or by scratching with a small hypodermic needle inserted through the wall of the appendix into the lumen. All these rabbits developed acute appendicitis and died within three days, except one which died after twelve days. This last one had a large abscess round the appendix. In two cases the appendix had perforated. *Figs. 536 and 537* show two appendices acutely inflamed; *Figs. 538 and 539* show the two perforated

FIG 536—Acute appendicitis. The animal died two days after ligation of the root of the appendix and crushing of the appendicular mucous membrane.

FIG 537—Acute appendicitis. The animal died twelve days after ligation of the root of the appendix, the mucous membrane of which had been scratched with a hypodermic needle. At autopsy the appendix was surrounded by an abscess.



appendices. The bacteria in the content of the appendix and peritoneum were similar to those seen in the normal appendix. Reverting to the two rabbits in the previous series which developed acute appendicitis, it seems reasonable to suppose that there was some lesion of the mucous membrane, either pre-existing, or more probably accidentally imposed at the time of operation. All the rabbits the mucous membrane of whose appendix was purposely damaged

died from acute appendicitis; whereas only two out of ten whose appendicular mucous membrane was not wittingly damaged developed appendicitis.

Obstruction of the Lumen of the Appendix by Foreign Body.—As the method of tying a ligature round the root of the appendix to produce obstruction seemed rather crude and unnatural, an attempt was made to produce the

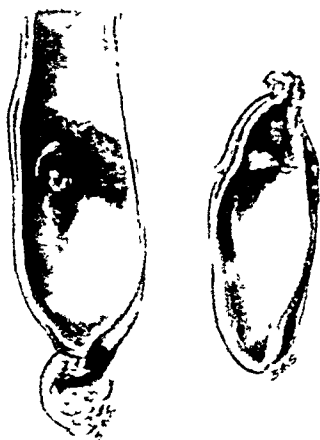


FIG 538—Acute appendicitis with perforation. The animal died thirty-six hours after ligation of the root of the appendix and crushing of the appendicular mucous membrane.

FIG 539—Acute appendicitis with perforation. The animal died two days after ligation of the root of the appendix and crushing of the appendicular mucous membrane.

obstruction from within. Foreign bodies were introduced into the lumen of the appendix through a small hole in the cæcum. This hole was closed with catgut and over-sewn. At first small pieces of glass tubing sealed at both ends and about 1 cm. long were introduced. The animals were killed three days later, and in all three cases the foreign body had disappeared from the appendix. There was no inflammation. As a substitute for

the glass bead, a cotton-wool plug was introduced, by means of a piece of glass tubing with a ramrod, through the cæcum into the appendix. This was done in ten cases. In none of these did any inflammation of the appendix follow. They were killed between five and ten days after operation. In three cases, one of which is shown in *Fig. 540*, the cotton-wool plug was still in the appendix; in the other seven it had disappeared.



FIG. 540.—A cotton-wool plug in the appendix inserted through a hole in the cæcum. The animal was killed six days after operation. There is no inflammation of the appendix.

Obstruction of the Lumen by Foreign Body and Damage to the Mucous Membrane of the Appendix.—If, as appears likely from the previous experiments, acute appendicitis in the rabbit follows on occlusion of the lumen when the mucous membrane is damaged, the same result should follow when the lumen is obstructed from within by a foreign body, if also the mucous membrane is damaged. In this series six rabbits were used. Three of these developed acute appendicitis and died. At autopsy the cotton-wool plug was in the appendix, which was acutely inflamed. The other three developed no signs of illness. When they were killed ten days later the foreign body had disappeared from the appendix.

DISCUSSION

The results of these experimental lesions are summarized in the table on p. 771.

Throughout these experiments, the criterion of judging acute appendicitis has been the death of the animal with an acute inflammation of the appendix and peritoneal cavity. For this reason no report on histological findings has been recorded here. In the event of survival the appendix has been examined for inflammation after a varying interval after operation.

Analysis of the experiments shows that the only experimental procedure which consistently produced acute appendicitis was obstruction to the lumen of the appendix combined with damage to the mucous membrane. It is immaterial whether the obstruction is by ligature outside the appendix or by a foreign body inserted into the lumen, provided that the foreign body remains in position. The fact that a foreign body placed in the appendix frequently disappears from that position led to a study of the physiological movements of the rabbit's appendix. The results of this work will be published later.

In order to correlate this experimental work with human appendicitis, it must be stressed that the human appendix is apparently subject to frequent inflammatory attacks which spontaneously terminate in fibrosis (Aschoff²). On this aspect of the disease the experimental work carried out sheds no light. On the other hand, there is a form of appendicitis in which the inflammation is progressive and, in the absence of surgical interference, leads to peritonitis and death. This form of the disease has been described by Wilkie,¹² who calls it "acute appendicular obstruction". The experiments here reported suggest that appendicular obstruction alone does not lead to appendicitis in the otherwise normal appendix. It is necessary that there shall be in addition a lesion of the mucous membrane. This

lesion need not be a gross one. A scratch with a fine hypodermic needle is sufficient to cause acute inflammation under experimental conditions. It seems likely that the mucous membrane of the human appendix is subject to small lesions, which in the presence of obstruction to the lumen will give rise to acute inflammation. This suggestion is supported by Aschoff (1932), who describes foci of inflammation in the mucous membrane of early cases of appendicitis and attributes its origin to them.

SUMMARY OF RESULTS OF VARIOUS EXPERIMENTAL LESIONS
IN RABBIT'S APPENDIX.

EXPERIMENTAL LESION	NUMBER OF RABBITS	RESULT		
		No Inflamma- tion of Appendix	Mucocoele	Acute Appendicitis
Injection of bacteria	12	12	0	0
Damage to mucous mem- brane	5	5	0	0
Damage to mucous mem- brane and injection of bacteria	5	5	0	0
Ligature of blood-vessels	3	3	0	0
Ligature of meso-appendix	3	3	0	0
Ligature of blood-vessels and meso-appendix	4	0	0	4
Kinking of appendix ..	4	4	0	0
Occlusion of lumen by ligature including blood- vessels .	4	4	0	0
Occlusion of lumen by ligature not tying blood- vessels	10	8	8	2
Occlusion of lumen by ligature and damage to mucous membrane	14	0	0	14
Obstruction to lumen by foreign body .	13	13	0	0
Obstruction to lumen by foreign body and damage to mucous membrane	6	3	0	3

SUMMARY

1. Experiments to determine the ætiology of acute appendicitis are described.
2. Injection of bacteria, either intravenously or directly into the appendix, isolated in most cases from inflamed human appendices, in no case caused appendicitis.

3. Gangrenous appendicitis and death followed ligature of the appendicular blood-vessels and meso-appendix.

4. Obstruction to the lumen of the appendix does not, in the rabbit, cause appendicitis. Such a procedure will, under certain circumstances, cause a mucocele of the appendix.

5. Obstruction of the lumen of the appendix, when the mucous membrane is damaged, is always followed by acute inflammation of the appendix and death of the animal. It is immaterial whether the obstruction is produced by ligature or by a foreign body.

REFERENCES

- ¹ MUIR, R., *Text-book of Pathology*, 1936. London: Edward Arnold & Co.
- ² ASCHOFF, L., *Appendicitis*, 1932. London: Constable & Co.
- ³ ADRIAN, C., *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1901, vii, 407.
- ⁴ POYNTON, F. J., and PAINE, A., *Proc. Roy. Soc. Med.*, 1911, v, 18.
- ⁵ ROSENOW, E. C., *Jour. of Infect. Dis.*, 1915, xvi, 240.
- ⁶ ROSENOW, E. C., *Surg., Gynecol. and Obst.*, 1921, xxxiii, 19.
- ⁷ HILGERMANN, R., and POHL, W., *Arch. f. klin. Chir.*, 1929, cliv, 248.
- ⁸ WEINBERG, M., ET AL., *Ann. de l'Inst. Pasteur*, 1928, xlii, 1167.
- ⁹ HEILE, *Münch. med. Woch.*, 1925, lxxii, 209.
- ¹⁰ WILLIAMS, B. W., and BOGGON, R. H., *Lancet*, 1934, i, 9.
- ¹¹ MORI, M., *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, xii, 639.
- ¹² WILKIE, D. P. D., *Brit. Med. Jour.*, 1931, i, 253.

INTESTINAL STRANGULATION: THE HISTAMINE CONTENT OF THE PERITONEAL TRANSUDATE FROM STRANGULATED INTESTINAL LOOPS

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INTRODUCTION

It has frequently been established that if a strangulated intestinal loop be isolated from the general peritoneal cavity by a rubber bag enclosing it (as first suggested by Foster and Hausler⁵), the blood-stained transudate which soon surrounds the loop, within the bag, develops toxic properties in a few hours. Holt⁷ confirmed the toxicity of this transudate, and showed that the lethal effect of injection of the transudate was lost after dialysis. He further concluded, by a study of the rate of passage of intestinal content through the wall of strangulated bowel, that the toxic material was derived from the wall and not from the lumen of the intestine. Knight and Slome⁸ demonstrated that the peritoneal transudate from completely strangulated bowel loops, if injected into other animals, produced a fall in blood-pressure.

One of us has shown elsewhere,^{1, 2} that at least two of the chemical constituents of the blood-stained transudate from strangulated intestinal loops produce toxic effects when injected into other animals. One of these appears to be a higher protein of the euglobulin class, or at least is precipitated with the euglobulin fraction of the transudate by one-third saturation with ammonium sulphate. The other toxic element of the transudate was diffusible, and killed young guinea-pigs so rapidly, with respiratory embarrassment, that the presence of considerable quantities of histamine was suspected. This histamine-like effect was a property of the toxic transudate of loops strangulated for some hours. It was not obtained from the protein-free filtrate of the non-toxic transudate from early strangulated loops.

The present paper describes a series of histamine determinations performed upon the peritoneal transudate from strangulated loops of cat intestine.

EXPERIMENTAL TECHNIQUE AND RESULTS

Collection of Material.—An 8-in. loop of the lowest ileum of an adult cat was inserted within a rubber balloon after the method of Foster and Hausler, and strangulated by a silk ligature tied around the neck of the balloon and the mesentery of the isolated segment, until the pulse of the artery of supply to the enclosed loop was just palpable through the balloon wall (*Fig. 541*). The balloon was returned to the abdomen and the animal allowed to recover. After a varying period of time the animal was killed, and from 15 to 50 c.c. of blood-stained fluid were recovered from the balloon. The bowel in each of the experiments remained unruptured. It was invariably deeply cyanosed or gangrenous at the time of death.

Method of Extraction of Histamine.—The technique applied by Gaddum and Barsoum⁶ to cat's blood was found equally applicable to the blood-stained transudate, and was adhered to in detail.

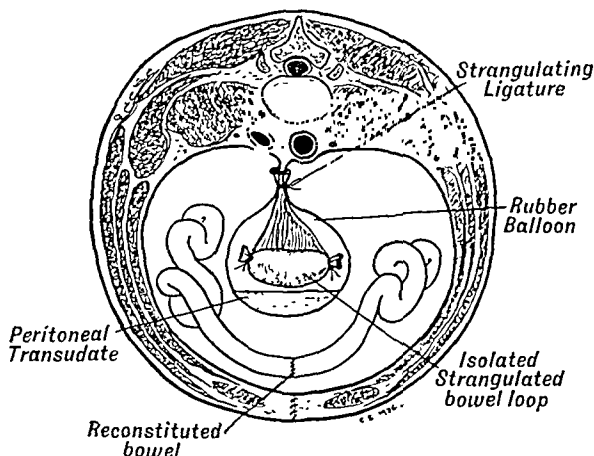


FIG. 541.—Cross-section of cat abdomen demonstrating, within the balloon, the transudate used for histamine determination. The strangulated isolated bowel loop from which the transudate derives, and the strangulating ligature around the balloon neck and mesentery, are also indicated. The bowel has been reconstituted. (Method of Foster and Hausler.)

Method of Histamine Estimation.—The extracts were tested, against known concentrations of histamine acid-phosphate, upon the blood-pressure of the atropinized cat, upon rat uterus, and upon the virgin uterus and ileum of guinea-pigs.

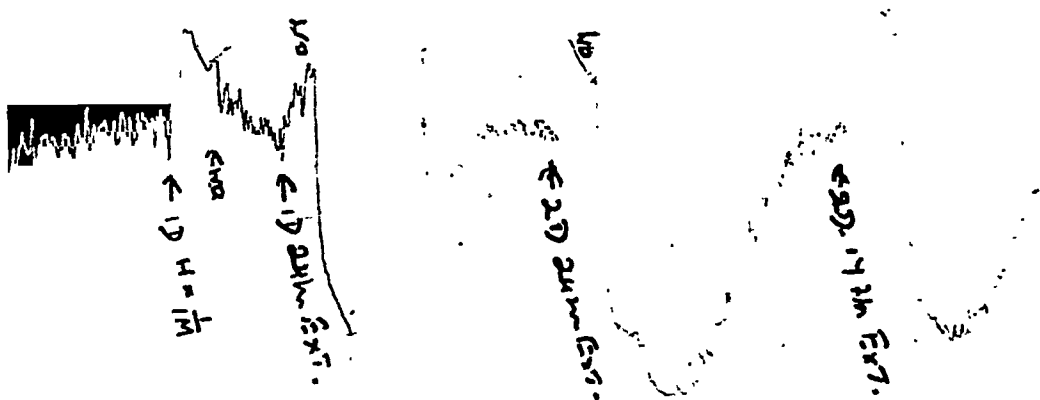


FIG. 542.—Guinea-pig ileum in Ringer-Locke. Extract = transudate concentrated 4 times. At 1st arrow, histamine added to concentration of 1-1,000,000 in bath. At 2nd, 24-hour extract added to concentration of 1-400. At 3rd, 24-hour extract added to concentration of 1-200. At 4th, 17-hour extract added to concentration of 1-200. (The depression of lever following each contraction is due to washing out bath.)

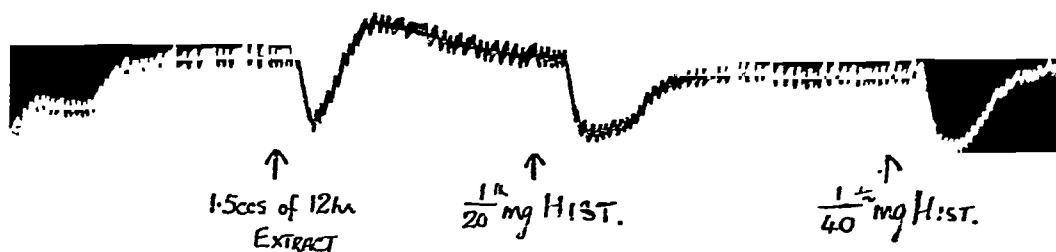


FIG. 543.—Blood-pressure of atropinized cat. At 1st arrow, 1.5 c.c. of extract of 12-hour strangulated loop transudate I.V. At 2nd, $\frac{1}{20}$ mg. histamine I.V. At 3rd, $\frac{1}{40}$ mg. histamine I.V.

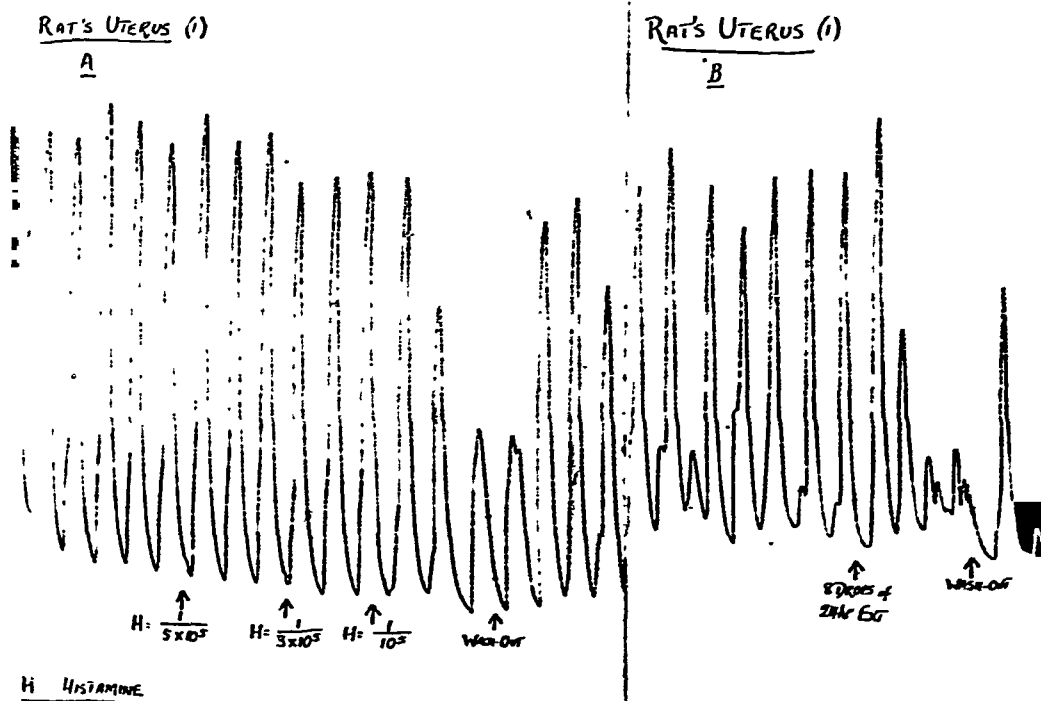


FIG. 544.—Rat uterus in Ringer-Locke. At 1st arrow, histamine added to concentration of 1-500,000 in bath. At 2nd, histamine added to 1-300,000. At 3rd, histamine added to 1-100,000. Wash out. At 4th, 24-hour extract added to concentration of 1-63. Wash out.

THE HISTAMINE CONTENT OF PERITONEAL TRANSUDATE OF STRANGULATED INTESTINE
AND OF STRANGULATED OMENTUM

TRANSUDATE EXTRACTED	BLOOD-PRESSURE OF ATROPINIZED CAT	VIRGIN GUINEA-PIG UTERUS	GUINEA-PIG ILEUM	RAT UTERUS	TOTAL VOLUME OF TRANSUDATE	TOTAL HISTAMINE CONTENT OF TRANSUDATE
Normal cat blood (Gaddum and Barsoum)	—	—	1/77,000,000	—	c.c. —	mg. —
6-hour strangulation of bowel	< 1/360,000 (1st Extract)	1/5,500,000 (2nd Extract)	1/8,000,000 (2nd Extract)	< 1/360,000 (1st Extract)	20	0.0025 to 0.004
12-hour strangulation of bowel	1/120,000 to 1/240,000	—	1/75,000 to 1/150,000	—	15	0.05 to 0.2
17-hour strangulation of bowel	< 1/20,000	—	1/20,000	1/16,700	50	2.5 to 3.0
24-hour strangulation of bowel	1/20,000 (1st Extract)	> 1/50,000 (1st Extract)	1/20,000 (1st Extract)	1/5,800 (2nd Extract)	25	0.5 to 4.0
27-hour strangulation of bowel	1/40,000 to 1/160,000	1/33,000	—	1/80,000	20	0.125 to 0.6
48-hour strangulation of bowel	1/40,000 to 1/160,000	1/33,000	—	1/40,000	30	0.2 to 0.9
42-hour strangulation of omentum	1/60,000	—	—	1/66,000	20	0.3
48-hour strangulation of omentum	< 1/160,000	—	—	< 1/360,000	7	< 0.03

The sign < means that the whole available quantity of extract has failed to elicit a histamine effect.
The sign > means that the effect of the extract has not been precisely measured, the paucity of material preventing a gradation in dosage. The figure given is calculated from that dose of histamine which gives the closest smaller response.

The results are expressed in the table as concentrations of histamine base. The method of extraction should be sufficient in itself to exclude the presence of other tissue-extract depressors. In a preliminary series of experiments, known solutions of adenosine and of acetylcholine were extracted by the same method and found to lose in the process their biological activity. It will be seen also that the histamine concentration in each separate sample varies little with the several biological preparations used for its determination. This is an added guarantee that histamine alone of the depressors has been present in our extracts, and that acetylcholine,

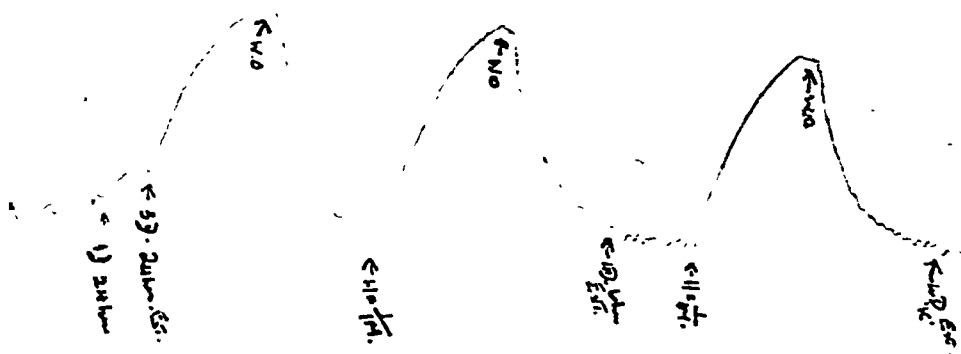


FIG. 545.—Virgin guinea-pig uterus in Ringer-Locke. At 1st arrow, 24-hour extract added to bath to concentration of 1-30. At 2nd, 24-hour extract added to 1-80. Wash out. At 3rd, histamine added to 1-1,000,000. Wash out. At 4th, 6-hour extract added to 1-40. At 5th, histamine added to 1-1,000,000. This tracing offers an excellent comparison of the histamine-like activity of the late extract, with the inactivity of the early extract.

adenosine, the P substance of Euler and Gaddum, and the depressor electrolytes have not been concerned. The main difficulty of the estimations lay in the limited amount of transudate available from any one strangulated animal. From the earlier strangulated loops only 4 or 5 c.c. of the extract could be prepared, and the results are less precise than they would be if an unlimited number of experiments permitted a close approximation of histamine effect and transudate effect. The shortage of material explains the use of 'less than' and 'more than' signs in the table. Figs. 542-545 show tracings obtained in the experiments.

DISCUSSION

It has repeatedly been shown that if a loop of small intestine of medium length be strangulated within a rubber bag to prevent peritoneal absorption, death is indefinitely delayed provided the lumen be reconstituted. In the absence of the rubber bag death occurs within two to five days, not necessarily from peritonitis, and without septicæmia. In strangulation of loops of medium length death is thus due to absorption of toxic peritoneal transudate derived from the strangulated loop: there is no drainage from the completely strangulated loop into the circulation except by the peritoneal route. The specific toxins which are present in the transudate, and which are responsible for death in medium-loop strangulation,

have not yet been identified. It is known at least that one of the toxic constituents is precipitated with the euglobulin fraction of the transudate and is not dialysable. Another is present in the protein-free filtrate of the transudate and is dialysable, and it kills young guinea-pigs in the same manner as histamine does. It is not surprising that histamine should be present in the transudate which bathes dead bowel—it would be surprising indeed if it were absent—but before any estimate is possible of the part histamine plays in the perhaps complex toxæmia of intestinal strangulation, the concentration of that substance in the peritoneal transudate must be determined.

The table of histamine concentrations indicates that the histamine content of the peritoneal transudate from a bowel loop increases progressively during the first twenty-four hours after establishment of complete venous strangulation. At the end of that period the histamine concentration of the transudate is from three thousand to fourteen thousand times greater than the histamine concentration which Gaddum has found in the normal blood of the cat. The histamine concentration of the transudate from strangulated loops seems to be maximal from seventeen to twenty-four hours after the induction of the strangulation, and does not appear to increase after that period.

The histamine content of the peritoneal transudate may amount, twenty-four hours after the establishment of strangulation, to 4 mg. Suddenly liberated into the blood-stream, this would be a large dose of histamine for a 3-kg. cat, but the total amount of histamine collected in a rubber bag during twenty-four hours of strangulation would, in the absence of the collecting bag, have been gradually absorbed by the peritoneum *pari passu* with its production. Dale³ has shown that the unanæsthetized cat is scarcely affected if 10 mg. per kilo body-weight is slowly infused intravenously over a period of fifteen minutes. There is, however, sufficient histamine in the transudate to explain its toxic effect upon guinea-pigs. Conditions, too, are present in intestinal strangulation which may affect the susceptibility of the organism to histamine. Venous strangulation is responsible for a diminution in the effective circulating blood-volume, and Dale has shown that anæmic animals give a severe response to histamine infusion. No claim is made that histamine should be regarded as the sole lethal agent in intestinal strangulation. The toxic euglobulin fraction of the transudate is at present under study, as are the other chemical constituents of the transudate—some of them perhaps present in too small a quantity to have of themselves a demonstrable toxic effect, but supporting each other in a cumulative effect upon the strangulated animal. There is sufficient histamine present in the transudate to bring it under suspicion as one of the factors in the toxæmia of intestinal strangulation.

The exact site of origin of the histamine of the peritoneal transudate has not been determined. The presence in the bowel of proteolytic bacteria in a pabulum rich in histidine would seem to offer an easy explanation of the ultimate source of the histamine, and it has been demonstrated by many methods^{1, 2, 4, 9} that strangulated loops of sterile bowel are innocuous. Some other factor than bacterial metabolism does, however, seem to be in part at least responsible. The peritoneal transudate from strangulated omentum, whose sterility was presumed though not actually confirmed, had after forty-two hours' strangulation a considerable content of histamine, though still considerably less than was present in bowel strangulations of shorter duration.

CONCLUSIONS

The histamine content of the peritoneal transudate from strangulated cat intestine increases until, after twenty-four hours of strangulation, it reaches a maximum of 1-5000 to 1-20,000.

The total content may amount to 4 mg.

While such a concentration is exceedingly high, it does not entitle histamine to be regarded as the sole lethal factor in intestinal strangulation.

The histamine in the peritoneal transudate is not of purely bacterial origin.

These experiments have been performed in the laboratories of the Departments of Surgery Research and of Pharmacology of Edinburgh University, under the guidance of Professor Sir David Wilkie and Professor A. J. Clark. Dr. H. Schild has assisted us to assess our results. One of us (I. A.) has been in receipt of a grant from the Medical Research Council; the other (W. K. H.) is a Vans Dunlop Scholar of Edinburgh University. The animal expenses have in part been borne by the Earl of Moray Endowment Fund of the University of Edinburgh.

REFERENCES

- ¹ AIRD, I., *Proc. Roy. Soc. Med.*, 1936, xxix, 991.
- ² AIRD, I., *Edin. Med. Jour.*, 1936, xliii, 375.
- ³ DALE, H. H., *Jour. Exper. Pathol.*, 1920, i, 103.
- ⁴ DRAGSTEDT, L. R., DRAGSTEDT, C. A., McCLINTOCK, J. T., and CHASE, C. S., *Jour. of Exper. Med.*, 1919, xxx, 109.
- ⁵ FOSTER, W. C., and HAUSLER, R. W., *Arch. of Internal Med.*, 1924, xxxiv, 697.
- ⁶ GADDUM, J. H., and BARSOUM, G. S., *Jour. of Physiol.*, 1935, lxxxv, 1.
- ⁷ HOLT, R. L., *Brit. Jour. Surg.*, 1934, xxi, 582.
- ⁸ KNIGHT, G. C., and SLOME, D., *Ibid.*, 1936, xxiii, 820.
- ⁹ MURPHY, F. T., and BROOKS, B., *Arch. of Internal Med.*, 1915, xv, 392.

EXPERIMENTAL OBSERVATIONS ON THE SPREAD OF CARCINOMA BY THE BLOOD-STREAM, WITH SPECIAL REFERENCE TO THE DIFFERENCE BETWEEN THE PORTAL AND SYSTEMIC ROUTES *

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INTRODUCTION

THE increasing success that is attending the treatment by surgery or irradiation of the local manifestations and lymphatic extensions of malignant disease in all parts of the body is emphasizing more and more the importance of spread by the blood-stream. For as more patients are cured of their local disease, more survive to develop blood-borne metastases, which so far remain as uncontrollable as ever. Until the day, therefore, when advances in knowledge provide a solution of the problem of malignant disease on the lines of prophylaxis and early diagnosis, the blood spread of tumours will be a subject of increasing importance.

Until recently studies of blood-borne metastasis had been practically confined to clinical and post-mortem material. But in recent years Foulds has shown that many of the problems lend themselves to experimental study in animals; and he has published interesting observations on the spread of a carcinomatous tumour of the rabbit following the introduction of tumour cells directly into a systemic vein. In particular, Foulds's work would seem to point to a possible resistant action on the part of the reticulo-endothelial system to blood-borne metastasis.

Scope of Present Work.—The present work was undertaken with the object of comparing experimentally the blood spread of malignant disease by the portal and systemic routes. It is well known from clinical experience that the incidence and distribution of blood-borne metastasis differs greatly in carcinoma in different situations: for example, it is very different in carcinoma of the large intestine and carcinoma of the breast. But it is impossible to say from clinical evidence alone to what extent these differences depend on the different characters of the tumours, and to what extent on the different anatomical relations in the two situations. Experimentally, this difficulty can be overcome by using the same tumour in both situations.

METHOD OF EXPERIMENT

The tumour used in the present experiments was the same as that used by Foulds—a carcinomatous tumour discovered by Brown and Pearce growing on the scrotum of the rabbit, and successfully transmitted by them to other rabbits by inoculation into various sites. The general technique and dosage were similar to those of Foulds. Briefly, a dilute cell emulsion in saline of the tumour was

* From the Bland-Sutton Institute of Pathology, Middlesex Hospital.

prepared, and one or two cubic centimetres were injected into the blood-stream of a series of rabbits. In the present experiments, the animals were divided into two groups, one receiving an injection into a systemic vein and the other the same dose into a radicle of the portal vein. In each experiment animals similar as far as possible in age, size, and breed were used, so that one group was a control for the other. For the systemic injection the ear vein was used; for the portal injection the animal was anæsthetized with ether, the abdomen opened, and the injection made into the main axial mesenteric vein of the small intestine. In some of the experiments (Nos. 9, 10, 14, 15, 16), in order to control the effect of the anæsthetic and operative procedure, the animals receiving an injection of tumour emulsion into a systemic vein were also anæsthetized, their abdomens opened, and an injection of saline given into the mesenteric vein. Surviving animals were killed usually about three to five weeks after the injection and a general examination was made of the tissues and organs, excluding the brain, spinal cord, and the interior of the bones, for deposits.

EXPERIMENTAL RESULTS

The results are expressed statistically in the table below. Some of the naked-eye and microscopical pictures found are illustrated in *Figs. 546-550*.

SUMMARY OF RESULTS OF INJECTION INTO THE BLOOD-STREAM

EXPERIMENT No.	TUMOUR INJECTED INTO PORTAL CIRCULATION		TUMOUR INJECTED INTO SYSTEMIC CIRCULATION	
	Number of Animals Injected	Number of Positive Results	Number of Animals Injected	Number of Positive Results
1	3	1	3	2
2	3	0	3	3
3	2	1	2	1
4	3	1	3	1
5	3	0	3	3
6	2	0	2	2
7	3	1	3	0
8	2	2	2	1
9	3	0	2	2
10	3	1	3	0
11	2	0	3	2
12	1	0	2	1
13	3	0	3	3
14	4	0	4	4
15	3	0	4	3
16	1	0	2	2
TOTAL	41	7	44	30

Frequency of Deposits in the Two Groups.—In all, 44 rabbits were injected by the portal route and 46 by the systemic. Three of the former and 2 of the latter died within ten days of the injection; and since in no case was any sign of a deposit noted before the tenth day, these cases may be excluded, leaving the results of 41 portal and 44 systemic injections for analysis. From the table it will be seen that out of the 41 animals receiving portal injections of the tumour emulsion, only

7 showed naked-eye evidence of tumour deposits, the remaining 34 being completely negative. Of the 44 animals receiving tumour emulsion into a systemic vein, 30 showed naked-eye deposits, and 14 were negative. Expressed in percentages, 17 per cent of the animals showed deposits in which the tumour was

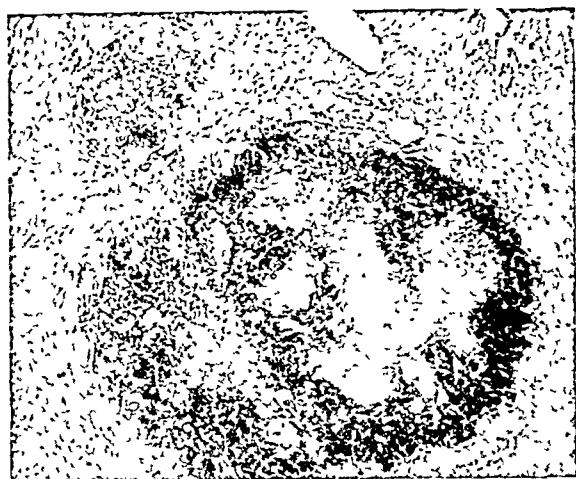


FIG. 546.—Low-power microphotograph of liver deposits to show the character of the tumour. The clear regions in the large nodule are areas of degeneration. ($\times 44$)



FIG. 547.—High-power microphotograph of liver deposits to show the character of the tumour. ($\times 120$)

injected into the portal circulation, and 68 per cent of those in which the injection was into the systemic venous circulation. Having regard to the controlled character of the experiments, it seems clear that the passage of the tumour cells into the portal vein is much less likely to give rise to deposits than a similar passage into a systemic vein.

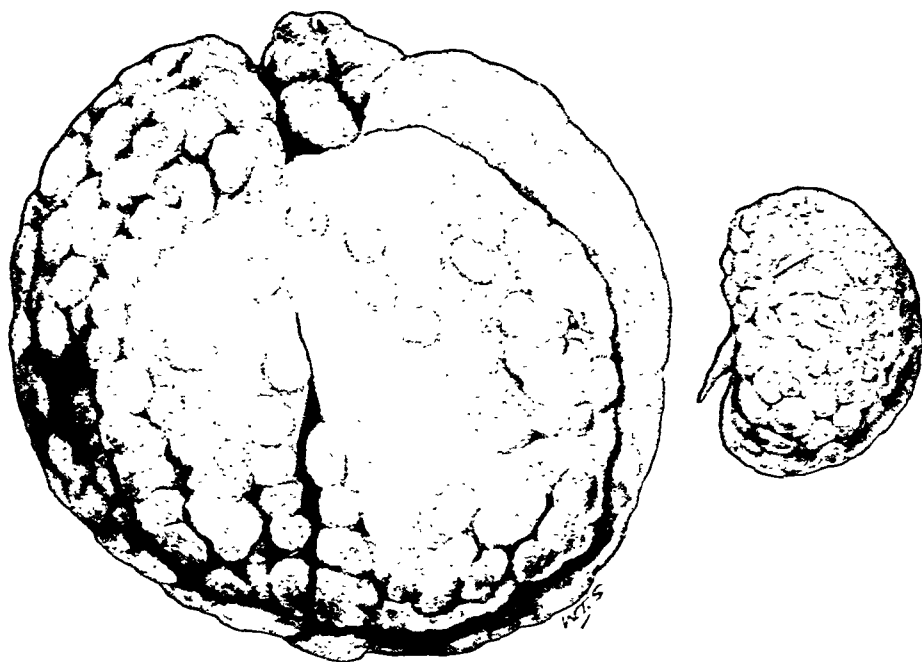


FIG. 548.—Drawing of the liver and kidney of a case in which the deposits were very numerous.

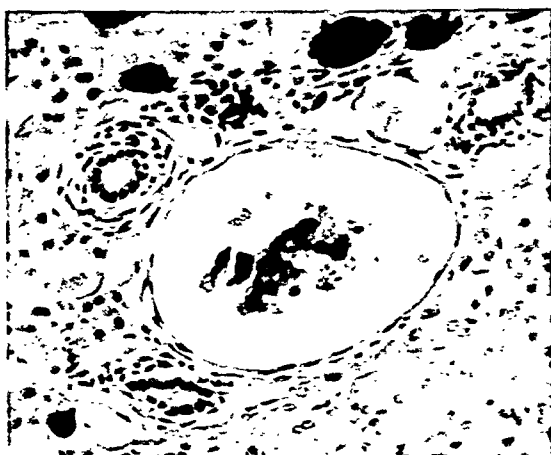


FIG. 549.—Microphotograph of a case in which numerous deposits were present in the liver, showing tumour cells in the portal vein. (This was taken from a case not included in the present experiments, in which indian ink was injected as well as tumour emulsion. The dark nodules in the surrounding liver are collections of indian ink.) ($\times 235$.)

This difference of incidence is also brought out on consideration of the individual experiments. Thus, while there are a few experiments in which the number of animals showing deposits following portal injection equalled (Experiments 3, 4), or even exceeded (Experiments 7, 8, 10), the number showing deposits following systemic injection, there is nothing comparable to the results illustrated in Experiments 2, 5, 6, 9, 13, 14, and 16, in which all of the animals injected by the systemic route showed deposits and none of the animals injected by the portal route.

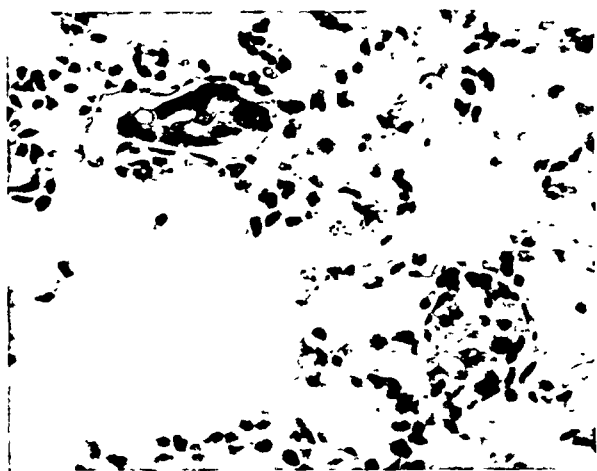


FIG. 550.—Two microscopical nodules of tumour cells in vessels in the lung, from a case in which the lung was free from growth to the naked eye. ($\times 272$.)

Distribution of Deposits.—The distribution of deposits following injection of the Brown-Pearce carcinoma into a systemic vein has been worked out by Foulds, who found that the principal organs affected were the kidney (90 per cent of positive cases), suprarenals (70 per cent), eyes (31 per cent), lungs (20 per cent), and liver (18 per cent). The distribution in the present series was roughly similar, with the exception of the less frequent involvement of the eyes. Thus, of the 30 positive cases, the kidneys were involved in 27, the liver in 13, the lungs in 11, and the eyes in 2. As regards the suprarenals, unfortunately in the earlier cases the appearance presented by early suprarenal deposits was not appreciated, so that many were undoubtedly missed. In the last 6 experiments, however, out of the total 18 cases suprarenal deposits were present in 12.

The small number of positive cases (7) following the intraportal injections makes definite conclusions on organ involvement difficult. Six out of the 7 showed deposits in the kidneys, 5 in the liver, and 2 in the lungs. In spite of the double barrier of the liver and lungs, the kidneys are, as in the systemic injections, the most frequently involved organs. The liver was involved relatively more frequently than after systemic injection, as would be expected, since all tumour cells had first to enter the liver; it is in fact rather surprising under these circumstances that the actual incidence of liver involvement following portal injection (5 out of 41 cases injected) was less than that following systemic injection (13 out of 44 cases injected). The number of deposits in the various organs in both groups of experiments varied

from a very large number, almost completely replacing the normal tissues, as in the case illustrated in *Fig. 548*, to a very few.

DISCUSSION

The experiments show that under controlled conditions tumour cells reaching the blood-stream by the portal vein give rise to metastatic deposits in markedly fewer cases than tumour cells entering through a systemic vein. This corresponds to the findings in human malignant disease, in which blood-borne metastases are much more frequent in tumours of the systemic territory, such as carcinoma of the breast and nævo-carcinoma, than in tumours of the portal territory, such as carcinoma of the large intestine. But in human malignant disease the facts are more complicated. For example, apart from differences in the type of carcinoma occurring in the two situations, a carcinoma of the breast rarely proves fatal from local extensions and complications, whereas a carcinoma of the large intestine frequently does; more cases of the former therefore survive to develop blood-borne metastases. Under experimental conditions the problem can be reduced to a simple level by having no primary growth and by injecting tumour cells directly into the blood-stream, and by using similar tumour in both situations. And the results of experiments of this type here recorded suggest that one of the factors responsible for the diminished incidence of blood-borne metastases in man, too, in tumours of the portal territory, as compared with tumours of the systemic venous territory, is the barrier of the liver.

The next question that arises is the manner in which the liver acts to some extent as a barrier to blood-borne metastases by the portal route. In the first place, pure mechanical factors must play a big part. Willis has calculated that most tumour emboli entering the blood-stream in cases of human malignant disease are of such a size that they must become arrested in the arterioles and capillaries of the first organ they meet; and a ground-up tumour emulsion such as is used experimentally is also composed essentially of aggregations of cells rather than single cells. Tumour emboli entering the portal vein have to pass through the capillary network of both the liver and the lungs before they reach the organs of the systemic circulation, whereas emboli entering a systemic vein have to pass only the capillary network of the lungs. The portal-borne emboli are thus doubly filtered, and for this reason alone metastatic deposits would be expected to be less. But there are some facts difficult of explanation on a purely mechanical basis. Thus, in the present experiments the liver showed deposits in 13 out of 44 cases injected by the systemic route, and 5 out of 41 injected by the portal route. On a purely mechanical basis, one would expect the incidence of cases with liver deposits to be much greater following portal injection, in which all the cells injected enter the liver, than following systemic venous injection, in which the liver has to share tumour emboli with all the other organs of the body. It is possible, of course, that some of the deposits in the liver following systemic injection represent secondary emboli from deposits in other organs such as the kidneys. But it is also possible, particularly in view of Foulds's work on the part played by the reticulo-endothelial system in the resistance to blood-borne metastasis, that the cells of this system in the liver play some part in the lower incidence of liver deposits following portal injection, and also in the lower total incidence of deposits.

The experiments also confirm the marked selective affinity of certain tumours for certain organs. Thus, in spite of the double barrier of the capillary network of the liver and the lungs, the kidney was the organ most frequently showing deposits after portal injection, just as after systemic injection. Thus anatomical vascular factors would seem to play a part quite subsidiary to this selective affinity.

In conclusion, the surgeon's interest in experimental blood-borne metastasis is primarily a practical one, and the questions he will at once ask are, first: Is the problem of blood-borne metastasis really any concern of the surgeon? and secondly: If so, is experimental study likely to afford any clue to its therapeutic control? The answer to the first question is that until the time arrives when malignant disease will be dealt with on the lines of the discovery and removal of the factors responsible for its development, the insidiousness of the disease will determine that a large proportion of the cases at the time they reach the surgeon will have already present blood-borne metastases, either obvious or latent. The subject is thus one of particular practical importance to surgeons. And as for therapeutic control, though the possibility of this at the moment seems remote, anything that is capable of giving further information on the problem is worthy of exploration.

SUMMARY

1. Controlled experiments are described in which the incidence and distribution of deposits are compared following the injection of a carcinomatous tumour of the rabbit into the portal circulation and into a systemic vein.
2. The number of animals showing deposits following portal injection is much less than following systemic injection.
3. The possible reasons for this difference and other related matters are discussed.

I have much pleasure in acknowledging my indebtedness to Professor J. McIntosh, Director of the Bland-Sutton Institute of Pathology, for his kindness in granting me the facilities of the Institute for the investigation.

REFERENCES

- FOULDS, L., *10th Scientific Report*, Internal Cancer Research Fund, 1932.
WILLIS, R. A., *The Spread of Tumours in the Human Body*, 1934. London: J. & A. Churchill.

THE BASIS OF TREATMENT OF VASOSPASTIC STATES OF THE EXTREMITIES: AN EXPERIMENTAL ANALYSIS IN MONKEYS.*

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* From the Laboratory of Physiology, Yale University School of Medicine. This work was completed while the writer was a Rockefeller Foundation Fellow. It was aided by a grant from the Rockefeller Foundation and the Research Funds of the Yale University School of Medicine. Part of the paper was read before the Harvey Cushing Society at the Mayo Clinic on May 16, 1936.

I. INTRODUCTION.

Sympathetic ganglionectomy is the usual surgical treatment of Raynaud's syndrome and other vasospastic states of the extremities. When the arm is affected, the conventional surgical procedure is to remove the second thoracic ganglion, the fused first thoracic and inferior cervical (stellate) ganglia, and sometimes the middle cervical ganglion, with the intervening portion of the sympathetic chain. This operation is usually known as cervico-thoracic ganglionectomy, and will be so named in this paper. Should the lower limb be affected the conventional operation consists in excision of the lumbar chain from the renal vein to the common iliac artery, and this usually includes the second, third, and fourth lumbar ganglia.

Experience has shown in the case of the foot that a good result may confidently be predicted. This is not so as regards the hand. In vasospastic disease of the upper limb the conventional operation is not always followed by adequate improvement, and frank gangrene has been known to occur shortly after operation. Telford,³¹ in a representative article (1934), writes, "All are agreed that whereas the result in the foot is almost always excellent, the result in the hand is often disappointing". Of 8 operations for Raynaud's syndrome of the foot he says, "All were completely satisfactory"; of 22 cases where the hand was affected, only 11 were "completely satisfactory". Jelsma and Spurling¹⁶ (1932) write, "We have treated 5 patients with typical Raynaud's syndrome during the past two years. The relief of symptoms in the lower extremities has been complete. . . . In only one of the five patients, however, have symptoms been completely relieved in the hands. One patient has lost all the fingers of the right hand, three of which became gangrenous following cervico-thoracic sympathectomy." Paterson Ross²⁸ gives statistics collected by the Association of Surgeons (1936): of 41 cases of Raynaud's syndrome of the hand, 29 were successful (17 of the 29 were followed up for less than a year after operation); of 20 cases affecting the feet, 1 only recurred, over two years after operation. These figures refer to cases treated by cervico-thoracic ganglionectomy or lumbar ganglionectomy as already defined.

The comparative failure of cervico-thoracic ganglionectomy to relieve attacks of vasospasm and to maintain relative vascular dilatation has been attributed to many factors. Regeneration has been suggested as a cause of 'recurrence', but it is highly improbable that regeneration can occur after ganglionectomy (across a synapse); furthermore, symptoms may recur less than a month after operation, long before regeneration could take place. Gask and Ross¹¹ believe that the discrepancy between the results obtained in the arm and leg is due to differences in the physiological activity of the vasomotor nerves and in the susceptibility of the vessels to cold. Woollard³⁵ also states (1935), "I think the better expectations of a good result from sympathectomy in the lower extremity are to be explained by the higher constrictor tonus of the lower extremity". Unwilling to admit an insuperable barrier to surgery such as so-called 'inherent vascular tone', some surgeons have believed their unsuccessful operations to be incomplete. Telford³¹ has subscribed (1934) to this view: "Whereas the lumbar operation insures complete sympathetic denervation of the leg and foot, cervico-dorsal ganglionectomy is not equally efficacious in achieving this object in the arm and hand". Kuntz¹⁷ inclines to the same idea (1934). Perhaps the staunchest supporters of this view are Adson and his associates. Simpson, Brown, and Adson²⁹ (1931) write, "Cervico-thoracic

sympathetic ganglionectomy occasionally fails to give complete relief because of failure to section all the sympathetic fibres. In our experience, lumbar sympathetic ganglionectomy is always completely successful."

Because these authors state their case more precisely and positively than do others, and because they offer a considerable body of evidence in its support, their evidence will be examined in some detail. The first part of the statement just quoted embodies conclusions reached from a study of 9 cases of Raynaud's syndrome of the hands fully reported in the same paper. Three of these cases were "complicated", having suffered periodic tissue loss from the finger tips. All 3 patients had attacks of vasospasm after operation, 2 of them within three weeks. The authors' statement, however, refers to uncomplicated cases without any evidence of organic narrowing of the vessels. Six cases fall into this group, but in 3 no operation is reported, and these will not be further considered. Of the 3 remaining patients, the first noticed attacks of pallor of the fingers when she handled cold or cool objects within two months of operation. At this time the patient was re-examined. When the hands were exposed to cold, "there were no changes in colour till the tips of the fingers

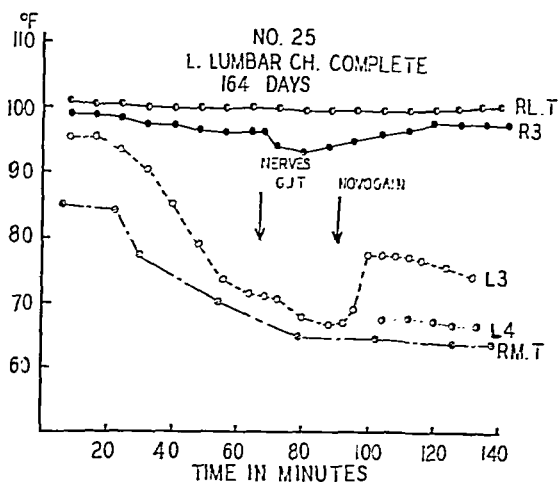


FIG. 551.—Monkey SS 25, five months after removal of left lumbosacral sympathetic chain. RL.T, Rectal temperature; RM.T, Room temperature; R3, Right third toe; L3 and L4, Left third and fourth toes. As the temperature of the room is reduced the sympathectomized toes cool. The left sciatic and femoral nerves are then exposed in the thigh, novocainized, and cut. The temperature of the corresponding sympathectomized toes does not alter. Twenty-five minutes later 2 per cent novocain is injected at the base of the third left toe, and the tip of the toe becomes warmer in 5 minutes. (For method of temperature recording, see p. 790 *et seq.*) It is not suggested that novocain exerts a specific dilator effect; the dilatation is probably due to a histamine-like substance released by the mild trauma of injection. (Doses of drugs used in this and all other experiments are expressed in cubic centimetres of solution per kilogram of body-weight.)

were massaged. A definite attack of pallor was then produced". Sympathetic denervation was proved to be complete by the sweating test. On these grounds the case is described as being "atypical". The second patient was free from all attacks at the end of a year. The third patient had an attack of blanching of two fingers of the right hand while in hospital after operation. When re-examined fourteen months later she was having frequent attacks in both hands, especially the right, and an attack could be provoked by immersing the hands in water at 15° C. The sweating test revealed that, "some fibres were still present in the right upper extremity, and to a slight extent in the left hand". With regard to this and similar cases the authors state, "By interrupting the fibres that have survived operation, further local anaesthetization can prevent signs of the disease". Their method of local anaesthetization was to block the ulnar nerve at the elbow and to inject novocain about the base of the fifth and fourth fingers. Under these conditions an attack could not be provoked in the anaesthetized fingers by immersing them in cold water. The authors conclude,

"Sympathetic ganglionectomy prevents manifestations of typical Raynaud's disease when all nerve-fibres are severed". Now Lewis has shown repeatedly that local anæsthetization of the ulnar nerve in such cases (uncomplicated as well as complicated) does not prevent attacks of vasospasm. Simpson, Brown, and Adson point out that the median and ulnar nerves normally communicate in the palm, and some uninterrupted vasomotor fibres may still reach the little finger when the ulnar nerve is blocked at a higher level. To remove this possibility they also anæsthetize the bases of the fourth and fifth fingers. This test, however, seems to be open to objection, as the following experiment carried out in the course of the present study shows:—

The sympathetic chain on one side was removed from the renal vein to the lower end of the sacrum in the monkey. When ample time had elapsed for degeneration of all the post-ganglionic fibres, the test was made (*Fig. 551*). The affected limb was allowed to cool, and the sciatic and femoral nerves were then divided in the thigh to make certain that no vasomotor fibres remained. No rise of temperature occurred. Some thirty minutes later novocain was injected about the base of the third toe. The temperature of this toe increased by 10° F. in ten minutes, and it remained relatively warm. The temperature of the fourth toe did not change, remaining just above that of the room. That is to say, local injection of novocain into a part long since deprived of all known vasomotor fibres is followed by a moderate degree of vasodilation which persists for some time.

To sum up: Of the 9 patients reported in the paper under discussion, 6 are automatically excluded; of the 3 that remain, 1 is in favour of the authors' contention, 1 is opposed to it, and the third cannot be admitted as evidence.

The weight of evidence thus points to the conclusion that complete sympathetic denervation of the upper limb in cases of Raynaud's disease is no guarantee against 'recurrence'.

It is to James White,^{30, 33} of Boston, that we owe a satisfactory explanation of the discrepancy between results in the upper and lower limb. He showed that the denervated blood-vessels of the hand become very sensitive to adrenaline after cervico-thoracic ganglionectomy, whereas after the conventional lumbar ganglionectomy the vessels of the foot remain relatively insensitive. White also pointed out the significant fact that by these operations the post-ganglionic fibres to the hand are divided, while it is the pre-ganglionic fibres to the foot that are cut, the post-ganglionic fibres remaining intact.

II. METHODS OF STUDY

This study is an attempt to analyse the early and late effects of division of vasomotor fibres on the responses of limb vessels by means of skin-temperature records. Attention is focused on the important differences between the effects of pre- and post-ganglionic section of the vasomotor fibres, and on the phenomenon of adrenaline sensitization. Because the essential feature of Raynaud's syndrome is reduction in the rate of blood-flow in the extremities, and because this is the sole cause of serious complications such as gangrene, it appears to be sound practice to base a study of the treatment of the malady on skin-temperature records; for the temperature of a given area of skin is an index of the rate of blood-flow through it and the underlying tissues, and not merely a measure of vasodilation.

A. SKIN-TEMPERATURE DETERMINATIONS

Rhesus monkeys (*Macaca mulatta*) were used in all experiments. Temperature records were obtained as follows: The monkey lies on its belly in an insulated box (*Fig. 552*), the temperature of which may be kept constant within 1° F., or may be altered at will. The animal's hands from the wrists, or its feet from the ankles,

and its head, protrude through small holes cut in the box, and are therefore surrounded by air at the temperature of the room. A thermocouple (constantan-iron) is attached by a small piece of adhesive tape to the palmar surface of the tip of each forefinger or the plantar aspect of each third toe, as the case may be. A third thermocouple is inserted four to five centimetres into the rectum. The thermocouples are connected to a 'Micromax' recording potentiometer (Leeds and Northrup). Stable conditions have been obtained by the use of dial as a light

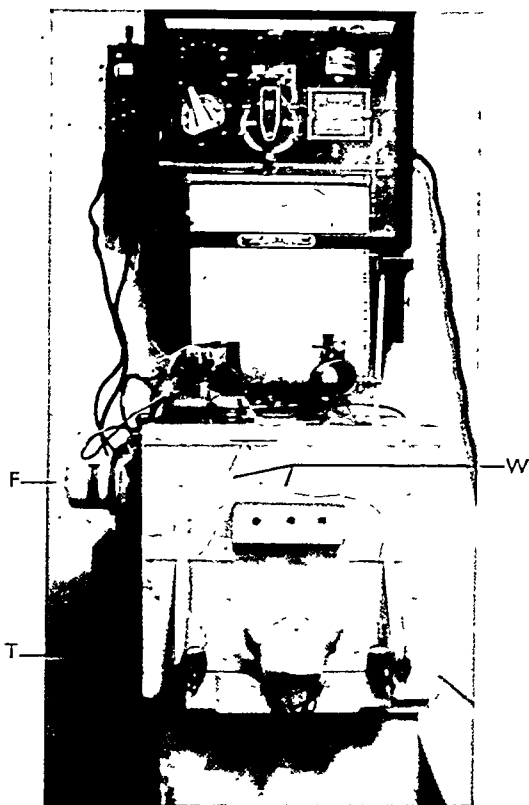


FIG. 552.—To show how temperature records were taken. Temperature recording machine above; below a monkey is shown in the box, with thermocouples attached by small strips of adhesive tape to the tips of the forefingers. W, Leads from thermocouples. The animal lies on a wooden grating which divides the upper compartment of the box from the smaller lower compartment. An electrically driven fan, F, circulates the air in the box by abstracting it from the top and blowing it back into the lower compartment down the tunnel, T. The fan contains an automatically controlled heater which warms the air. In the lower compartment is a coil of copper pipe (not shown), connected to a refrigerating system. By means of these heating and cooling arrangements, the temperature of the air in the box may be rapidly adjusted to any temperature from 60 to 160° F.

anæsthetic, 0.4 c.c. per kilogram of body-weight being given by intraperitoneal injection, and it is important to bear this in mind in the interpretation of the records here reproduced. However, there is good evidence that dial does not notably depress the activity of the autonomic system, at all events at subcortical levels (Grant,¹³ Cannon²⁵). Anæsthesia was rarely deep enough to abolish the corneal reflex.

Continuous temperature records lasted two to twenty hours, usually about five hours, and they were repeated at intervals up to eleven months after operation.

The normal rectal temperature of the monkey is about 101° F. A short period of exercise, for example chasing the animal about the room, will raise it 2 or 3° F.

B. ANATOMY

As it has been difficult to obtain necessary information concerning the anatomy of the sympathetic system of the rhesus monkey, several dissections have been made. These show that there are many variations in the detailed anatomy, but

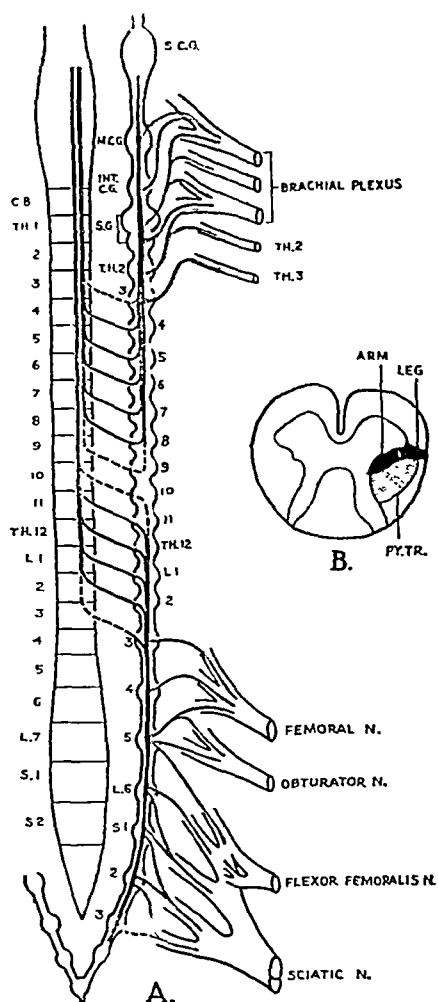


FIG. 553.—A.—Diagrammatic representation of outflow from the spinal cord of pre-ganglionic (red) and post-ganglionic (green) sympathetic vasomotor fibres to the limbs in the rhesus monkey (based on the researches of Langley¹⁸ and Gaskell¹⁹). Broken lines indicate inconstant fibres. S.C.G., Superior cervical ganglion; M.C.G., Middle cervical ganglion; INT. C.G., Intermediate cervical ganglion; S.G., Stellate ganglion; TH.2, second thoracic ganglion; L.1, First lumbar ganglion; S.1, First sacral ganglion. B.—To show position of the vasomotor fibres in the spinal cord, and their relation to the pyramidal tract, PY.TR. (After Foerster).

that the major features are as constant as they are in man, and that the monkey's sympathetic system as a whole closely resembles its human counterpart.

Fig. 553 represents diagrammatically the sympathetic vasomotor outflow to the extremities, and it embodies the brief description which follows of the relevant anatomy and the experimental surgical procedures.

The brachial plexus in the rhesus monkey arises from the same nerve-roots as in man, and it is similarly constituted. The most caudal grey ramus to the brachial plexus arises from the 2nd thoracic sympathetic ganglion and runs to the 1st thoracic nerve; grey rami to the 8th, 7th, 6th (and sometimes the 5th) cervical nerves arise from the stellate ganglion and constitute the major portion of the vertebral nerve. Smaller branches arise from the stellate ganglion, or the lower part of the cervical sympathetic chain, intermediate cervical ganglion, and middle cervical ganglion, and course laterally in front of the scalenus anticus to reach the brachial plexus, mainly in its upper part. The pre-ganglionic fibres to the upper limb arise mainly from the 4th or 3rd thoracic segment to the 9th (Langley¹⁸). It is possible that a few fibres arise also from the second segment. Most or all of the pre-ganglionic fibres to the eye arise not lower than the 1st thoracic segment. According to Foerster the vasoconstrictor fibres to the arm in man arise from the 2nd thoracic segment to the 6th or 7th.

There are seven pairs of lumbar nerves in the monkey, and usually six pairs of lumbar sympathetic ganglia. The sciatic nerve as found in man is represented in the monkey by a loose combination of the internal and external popliteal nerves below the greater sciatic notch, formed of branches from the 5th lumbar nerve to the 2nd or 3rd sacral nerve. These receive grey rami in rather irregular fashion from the corresponding sympathetic ganglia. Pre-ganglionic fibres to the lower limb arise from the 9th thoracic segment to the 2nd or 3rd lumbar segment. The femoral nerve arises from the 3rd, 4th, and 5th lumbar nerves, or from the 4th and 5th only, and the obturator nerve from the 4th and 5th lumbar nerves (Hartman and Strauss,¹⁵ and personal dissections).

C. OPERATIONS

The following operations have been most often employed in these studies:—

1. Upper Limb.—

a. Post-ganglionic Division of the vasomotor fibres consists in removal of the second thoracic ganglion, the fused first thoracic and inferior cervical ganglia, the middle cervical ganglion and the intervening parts of the sympathetic chain, and in division of all rami at a short distance from their attachment to the ganglia. The operation is carried out from the front, and it is rendered simple by dislocating the inner end of the clavicle and retracting it downwards. The clavicle is easily stitched back into position, and the use of the limb is not impaired.

b. Pre-ganglionic Division is performed by removing the inner end of the third rib through a posterior incision, by identifying the easily accessible thoracic sympathetic chain, and by dividing it just above the third thoracic ganglion, whose grey and white rami are also cut. Regeneration is prevented by ligaturing the crushed ends of the divided thoracic chain and tying them back into the opened intercostal space, the upper end up and the lower end down. Pre-ganglionic division has also been done by intradural division of the 3rd to the 9th thoracic anterior roots, the corresponding posterior roots being left intact.

2. Lower Limb.—

a. Post-ganglionic Division is done by removing the lumbosacral sympathetic chain transperitoneally from the renal vein to the lower end of the sacrum. This includes all the sacral ganglia and all the lumbar ganglia except the first two.

b. Pre-ganglionic Division is done in the same way, except that the chain is removed only as far down as the level of the bifurcation of the aorta, and thus includes lumbar ganglia 2, 3, and 4 (the second ganglion may escape).

Post-ganglionic and pre-ganglionic sympathectomy are often referred to in the following pages as *denervation* and *decentralization* respectively.

III. EXPERIMENTAL FINDINGS

A. VASCULAR REACTIONS OF NORMALLY INNERVATED LIMBS

The temperature changes which occur in normal extremities on exposure to cold are shown in numerous figures. So far as we can determine, sympathectomy

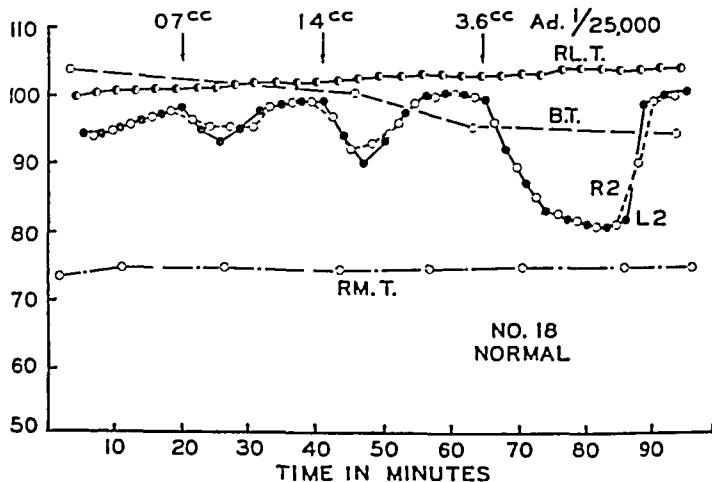


Fig 554—Monkey SS 18, unoperated animal. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show the action of adrenaline in normally innervated fingers. Three injections of 1-25,000 solution of adrenaline are given under conditions favouring vasodilatation. Each dose is followed by a fall in temperature of the fingers indicating vasoconstriction, proportional to the quantity of adrenaline injected. In this animal the cooling effect is a little less in the right forefinger than in the left. This may be ascribed to some unrecognized defect in experimental technique, or to a slight difference between the vessels of the hands in an apparently normal animal. Numerous tests of this kind have been made and we have not observed greater differences in normal bilaterally symmetrical parts than that shown here.

of one limb has no significant effect, temporary or permanent, on the vessels of the other limbs. Fig. 554 shows the reaction of the normal monkey to increasing doses of adrenaline. Injections were made into the temporalis muscle in this and all other experiments, and all doses are expressed in cubic centimetres of solution per kilogram of body-weight. The degree and duration of vasoconstriction following injection of adrenaline is seen to be proportional to the dose. In normally innervated limbs injection of a small quantity of adrenaline is often followed by a rise in temperature, due to vasodilatation (Fig. 555). Though the rise is not great, it represents a considerable increase in blood-flow, for the temperature of the fingers is already not far below that of the blood. Such vasodilatation is only seen when the part is already warm; it has not been found to occur when the

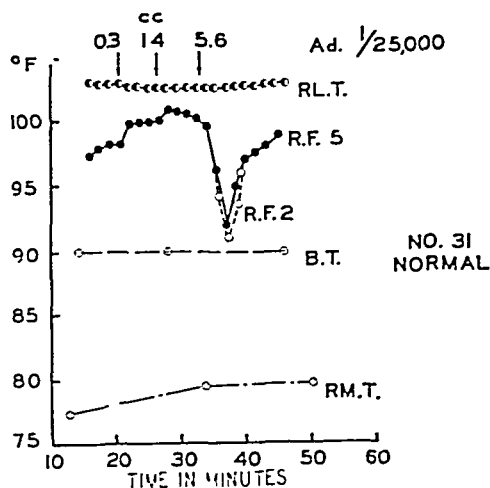


FIG. 555—Monkey SS 31, unoperated animal. R F. 2, Right forefinger; R F. 5, Right little finger; RL. T., Rectal temperature, B T., Box temperature, RM. T., Room temperature. To show that small doses of adrenaline in the normal animal may be followed by vasodilatation. In this animal a large quantity is required to produce vasoconstriction. The temperature of the forefinger is the same as that of the little finger except where shown.

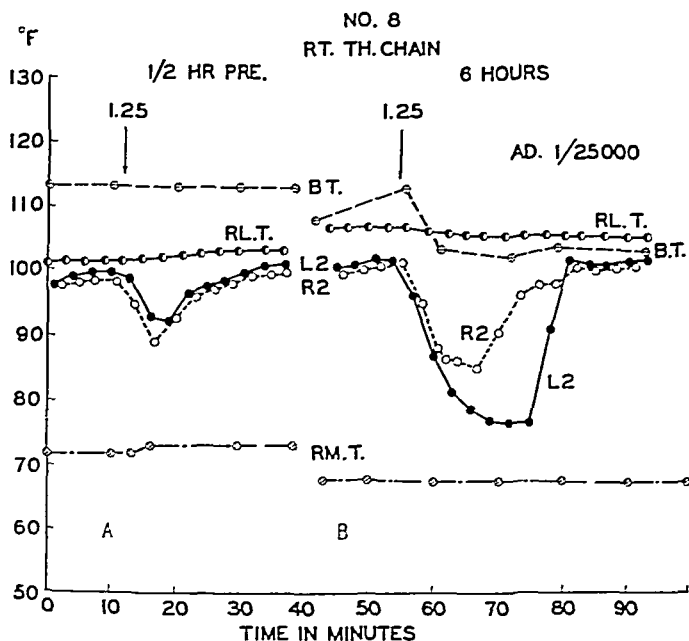


FIG. 556—A, B—Monkey, SS 8, division of right thoracic chain at third ganglion. R2 and L2, Right and left forefingers; RL. T., Rectal temperature, B. T., Box temperature, RM. T., Room temperature. Record A was obtained just before operation. The animal was hard hit by the operation, which was unusually severe. Record B, taken 6 hours after the wound was closed, illustrates two points: the first is that one of the immediate effects of sympathectomy is to reduce somewhat the constrictor effect of adrenaline on sympathetomized vessels compared with those of the normal side; the second is that an animal in poor condition becomes generally sensitized to adrenaline—a dose of adrenaline equal to that given to this animal before operation afterwards produces a distinctly greater and more prolonged fall in temperature on both sides.

fingers and toes are cold, no matter how small the dose of adrenaline employed. Having determined the dose of adrenaline which was followed by a moderate degree of vasoconstriction, ergotoxine was injected in a group of five monkeys and the same doses of adrenaline repeated. In each animal the dose after ergotoxine was followed by vasodilatation (*Fig. 568*). This phenomenon of "adrenaline reversal", first described by Dale,⁶ affords further evidence in favour of the presence of vasodilator fibres to the peripheral cutaneous vessels in the monkey.

Figs. 554 and 555 show that there is some variation in the degree of constrictor response of the vessels of healthy animals to adrenaline. Should an animal be in poor health, it is interesting to observe that it will show a considerable degree of general vascular sensitization. An animal ill from pulmonary tuberculosis, or in poor general condition, or suffering from the effects of several doses of anæsthetic or from a severe operation, will show an over-reaction to adrenaline (*Fig. 556*). The pallor of ill health in human beings may be of the same nature. Grant has suggested that this sensitization to injected adrenaline is only apparent, and that part of the constrictor effect is to be attributed to an adrenaline-like substance produced in the body in ill health.

B. IMMEDIATE EFFECTS OF SYMPATHECTOMY

Division of the vasomotor fibres to an extremity is followed within a few minutes by profound vasodilatation which is very difficult to overcome by cooling. This is true whether the lesion is made by dividing the peripheral nerves, by hemisection of the spinal cord, or by interrupting the vasomotor fibres at any intermediate level. The vasodilatation is perhaps most intense when the lesion is post-ganglionic, but the difference is small. *Fig. 557* shows that shortly after the animal left the operating table the temperature of the sympathectomized leg was only about 4° F. below the rectal temperature, although this was 5° F. subnormal, whereas the normal foot was rapidly cooling. When the room temperature was quickly reduced almost to freezing-point the normal toes assumed a temperature barely higher than that of the surrounding air. The sympathectomized toes also began to cool, but their temperature soon levelled off at about 75° F. It is clear that at least part of this fall on the operated side may be accounted for directly by increased heat loss due to the steeper gradient between the temperature of the body and that of the air. The rate of heat loss from the toes is directly proportional to the difference in temperature of the blood entering the toes and that of the surrounding air, assuming that the rate of blood-flow is constant. Before the room is cooled, the ratio between the difference in temperature of the blood and the air, and the blood and surface of the toe is, in this case :

$$(96^{\circ} - 76^{\circ}) : (96^{\circ} - 92^{\circ}) = 20 : 4 = 5 : 1.$$

After the room is cooled the ratio is :

$$(95^{\circ} - 35^{\circ}) : (95^{\circ} - 75^{\circ}) = 60 : 20 = 3 : 1.$$

That is to say, the rate of heat loss is either increased disproportionately to the fall in temperature of the room, or the rate of blood-flow is reduced by some degree of vasoconstriction. In these experiments, the room was cooled by opening the door and window, allowing a cold draught to blow directly on the exposed feet. The following experiment affords evidence that the draught alone probably accounts

for the disproportionate heat loss. The leg of a recently dead animal was amputated at the knee. From the middle toe the flexor tendons were removed and a small electric heater consisting of a small glass tube containing a coil of platinum wire was placed in the tendon-sheath, a thermocouple was fixed to the surface of the tube, and the skin was closed. A second thermocouple was applied to the skin directly over the first, and a third was arranged in the air one inch away

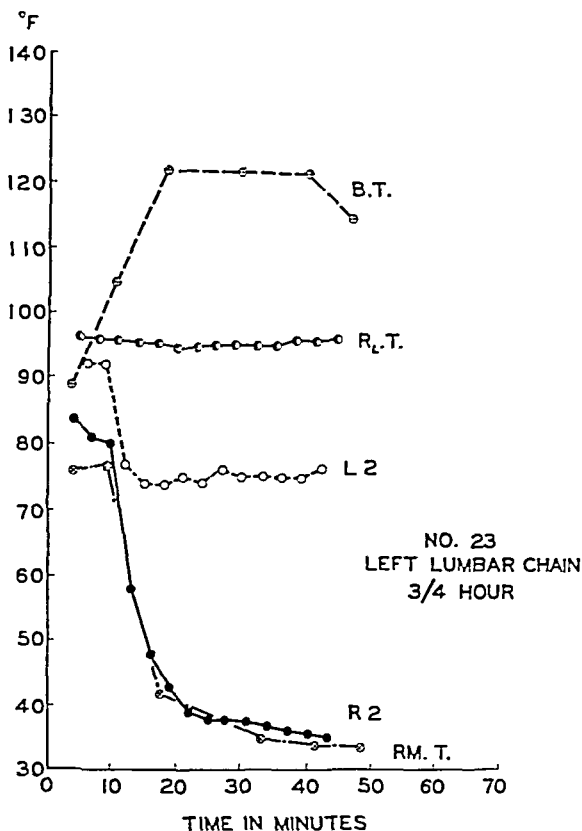


Fig. 557.—Monkey SS 23, three-quarters of an hour after removal of left lumbosacral chain. R2 and L2, Right and left second toes; R.L. T., Rectal temperature; B. T., Box temperature; R.M. T., Room temperature. The newly sympathetomized toes remain warm though the rectal temperature is only 96° F. and room temperature is reduced almost to freezing-point. The room was cooled by opening the door and window, admitting an icy draught.

from the toe. Fig. 558 shows the temperature differences between the skin, the heater (crudely representing the blood-stream), and the room air. The supercooling effect of draught is conspicuous. Ratios calculated similarly to those already given are as follows: Fig. 558A, no draught, 5:1; Fig. 558B, moderate draught, 2.8:1; and Fig. 558C, strong draught, 1.5:1. These figures demonstrate that the disproportionate fall in temperature of the newly sympathetomized part in Fig. 557 may be accounted for by the cold draught. If any vasoconstriction occurs it is very slight. (Full dilatation may persist in the newly sympathetomized part when the rectal temperature is as low as 86° F.)

It may be concluded that the immediate effect of sympathectomy is to abolish the vasoconstrictor response to external cold and to reduction in body temperature within very wide limits.

Though sympathectomy has so remarkable an immediate effect on the reaction of vessels to cold, their constrictor response to adrenaline shows little change. During the first twenty-four hours after operation the vasoconstrictor reaction is, nevertheless, rather less on the sympathectomized side than on the normal side (*Fig. 556 A, B*). This is so after peripheral nerve division, sympathetic ganglionectomy, pre-ganglionic trunk division, and hemisection of the spinal cord.

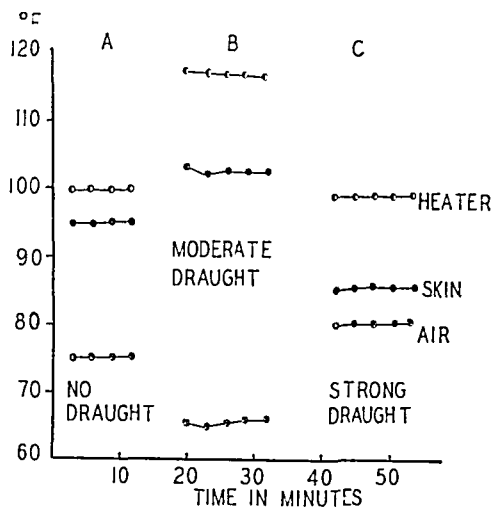


FIG. 558.—Amputated foot. A small electric heater is placed in the tendon-sheath of a toe from which the flexor tendons have been removed. Having shown that the temperature difference between the heater (crudely representing the warm circulating blood) and the overlying skin is directly proportional to the temperature of the surrounding air, the experiment here illustrated was done. The chart indicates the notable cooling effect of draught.

C. LATER EFFECTS OF SYMPATHECTOMY

1. Post-ganglionic Sympathectomy.—There is a considerable and clinically important difference between the later effects of pre- and post-ganglionic lesions. The condition following sympathetic denervation, typified by the conventional operation for vasospasm of the upper limb, will be described first. *Fig. 559* shows the temperature changes of the forefingers of an animal four months after left cervico-thoracic sympathetic ganglionectomy. Vasomotor activity has returned to a considerable degree in this animal, and the changes in tone of the sympathectomized vessels are unusually brisk. When the temperature of the room is reduced from 72° F. to 41° F., constriction of the normal and sympathectomized vessels results to an almost equal degree. When the room is warmed and the rectal temperature rises a little, dilatation of the sympathectomized vessels is delayed and takes place more slowly than on the normal side. Though most animals subjected to cervico-thoracic ganglionectomy react to cold in a manner comparable to this, there is some individual variation, and a considerable degree of vasodilatation may persist in the face of cold sufficient to cause abrupt vasoconstriction

in the normal limbs. In our experience this is unusual, only one monkey in twelve showing what may be termed a good degree of chronic vasodilatation (*Fig. 571*, first part of chart). Moreover, it seems important to realize that denervated vessels may react to cold even more intensely than those of the normal side (*Fig. 560*). That is to say, cervico-thoracic sympathetic ganglionectomy sometimes induces in the normal animal a state comparable to that which the operation is designed to relieve in patients with peripheral vasospastic disease.

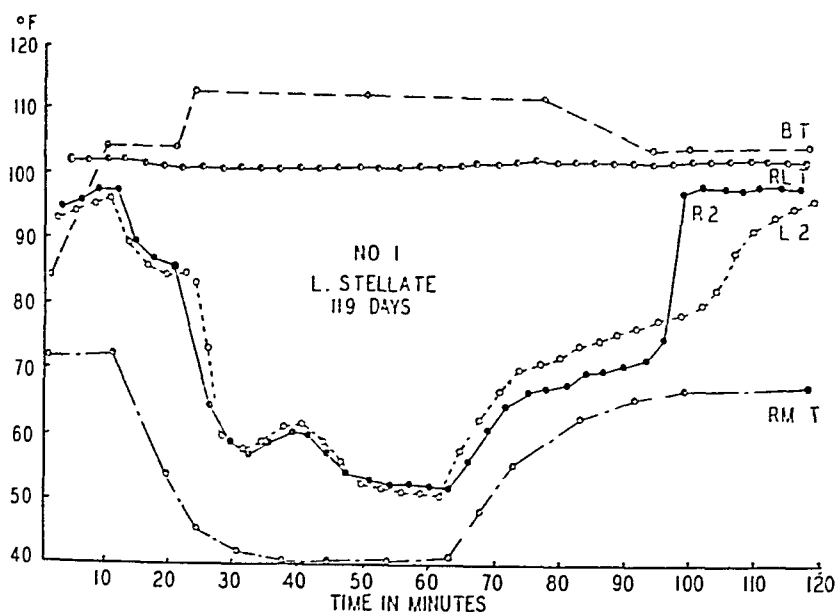


FIG. 559.—Monkey SS 1, four months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show the great recovery of vasomotor activity ('tone') in sympathetomized vessels, after post-ganglionic section of vasomotor fibres. At first the room is warm and conditions favour vasodilatation, and the sympathetomized finger is almost, but not quite, as warm as the corresponding normal finger. On cooling the room the fingers also cool, at first owing to increased heat loss. Ten minutes later there is an abrupt further fall in temperature, indicating that vasoconstriction has occurred. When the room is heated the sympathetomized fingers, at first a little warmer than their fellows, lag behind the normal side. The close parallel between the two sides seen in the first half of this chart is unusual, and is probably due to the rapidity and depth of the changes in room temperature. Temperature changes in sympathetomized limbs exposed to less abrupt and extensive alterations in temperature occur more slowly, as in the second half of the chart.

In two animals the post-ganglionic fibres to the lower limb were similarly cut by removing the lumbosacral chain from the renal vein to the lower end of the sacrum, and in both animals recovery of reactivity has been rather more rapid and complete than in most of the upper limb denervations (*Figs. 551 and 564*).

No attempt is made here to give a detailed description of the reactions of vessels that have lost their post-ganglionic fibres, since unrivalled accounts are available in the publications of Lewis,^{21, 22, 23} and Grant^{13, 14} and their associates.

2. Pre-ganglionic Sympathectomy.—The experiment on which *Fig. 561* is based showed that in the presence of moderate cold sufficient to cause nearly complete closure of the vessels of the normal fingers, evidenced by the close approximation of their temperature to that of the room, considerable vasodilation

persists on the decentralized side months after operation.* *Fig. 562* shows that a similar state of affairs holds after the corresponding operation in the lower limb

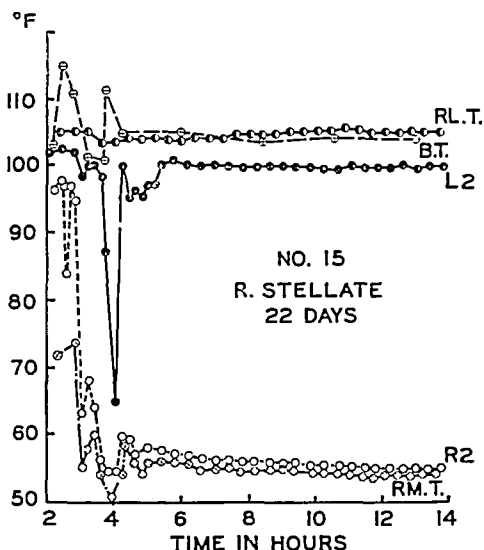
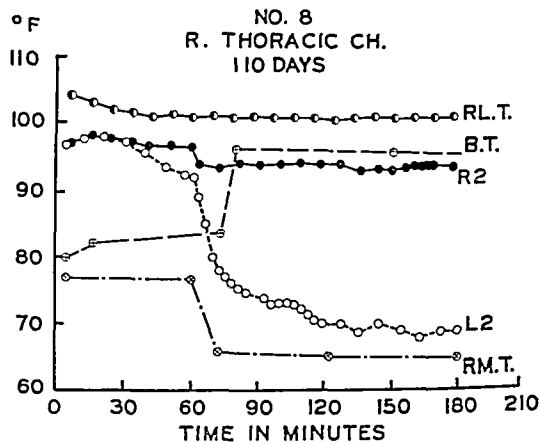


FIG. 560.—Monkey SS 15, twenty-two days after right cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show that a part deprived of its post-ganglionic nerve-supply may react to cold with abnormal readiness. Rectal temperature is kept above normal so that vasoconstrictor impulses are inhibited; the normal hand remains warm except when the room is supercooled for a short time. The sympathectomized fingers, warm at first, cool when the temperature of the room falls from 74° to 55° F, and they remain cold. Their temperature remained within 1° F. of the room temperature for fifteen hours. No signs of local nutritional disturbance could be detected subsequently.

(the conventional operation in man). The degree of vasodilatation under average conditions tends to be less than that present immediately after operation, and it is

FIG. 561.—Monkey SS 3, three and a half months after division of right thoracic chain; R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show chronic vasodilatation after pre-ganglionic division of vasomotor fibres, in contrast to *Figs. 559, 560*. The finger deprived of its pre-ganglionic supply (post-ganglionic fibres intact) remains warm throughout. The normal finger begins to cool when the rectal temperature is reduced, and further rapid cooling follows the fall in room temperature.



* Grant has shown that the 'tone' of vessels deprived of their pre- or post-ganglionic fibres is largely independent of reduction in body temperature, because vasoconstrictor impulses of central origin, arising in consequence of cooling of the blood, cannot reach the vessels. This is the probable explanation of the divergence in temperature between the normal and sympathectomized fingers in this chart (*Fig. 561*) during the first hour when the rectal temperature falls from 103° to 101° F. Under conditions such as those which hold during that time, relative vasodilatation may similarly persist after division of post-ganglionic fibres. But vessels whose post-ganglionic fibres are intact and whose pre-ganglionic fibres are cut are to a considerable extent unaffected by external cold also, as may be seen in the second half of *Fig. 561* (and in *Fig. 562*, where the rectal temperature remained at 103° to 104° F.); whereas vessels without post-ganglionic fibres constrict to a normal or supernormal degree under similar conditions (*Figs. 559, 560*).

more easily reduced by cold, yet it is considerable when compared with the normal limb or a limb subjected to post-ganglionic sympathectomy.

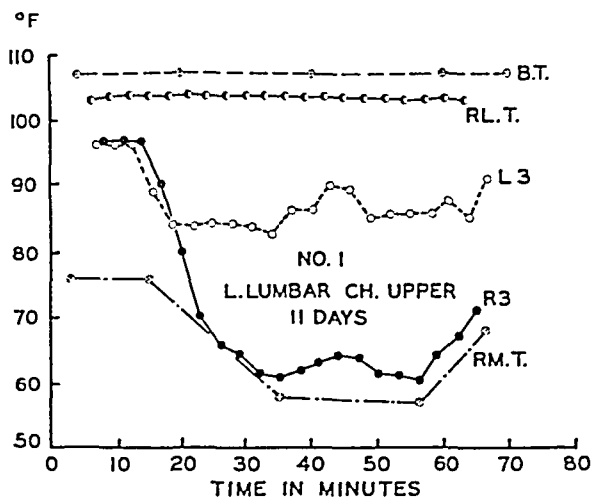


FIG. 562.—Monkey SS 1, eleven days after removal of left lumbar chain with ganglia 2, 3, and 4. R3 and L3, Right and left third toes; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show that vasodilatation persists after pre-ganglionic sympathectomy, as in Fig. 561. This is compatible with the condition observed in man years after the same type of operation.

3. Adrenaline Effects.—The reaction to adrenaline some time after cervico-thoracic ganglionectomy is illustrated in Fig. 563. The denervated part, its

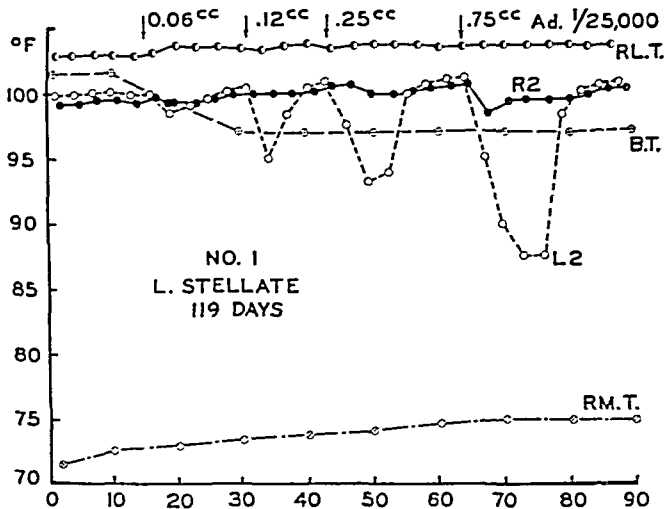


FIG. 563.—Monkey SS 1, four months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show adrenaline sensitization after post-ganglionic sympathectomy. The first injection of 0.06 c.c. 1-25,000 adrenaline per kilo of body-weight produces a minimal constrictor effect on the sympathectomized side and a slight dilator effect on the normal side; 0.75 c.c. (more than ten times the first dose) is required to produce a fall in temperature on the normal side.

post-ganglionic fibres having degenerated, is about ten times as sensitive as the control side. Fig. 564 shows that the corresponding operation in the lower limb

also leads to great adrenaline sensitization. *Fig. 565* illustrates a typical reaction to adrenaline after decentralization. The operated side is about three times as sensitive as the normal side. That is to say, decentralized vessels are a great deal

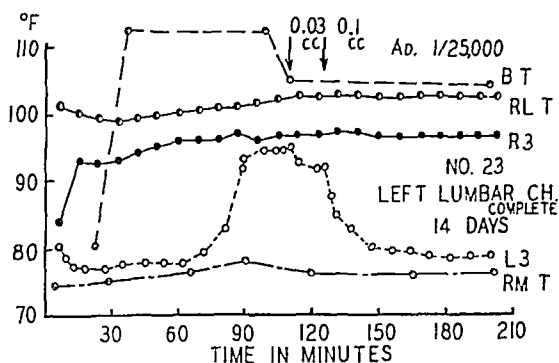


FIG. 564.—Monkey SS 23, fourteen days after removal of the lumbosacral chain. R3 and L3, Right and left third toes; RL T, Rectal temperature; BT, Box temperature; RM T, Room temperature. To show adrenaline sensitization and the great recovery of vasomotor activity or 'tone' after post-ganglionic sympathectomy in the leg. When the monkey, under dial, was placed in the box, the left foot was warm, the right cool. The record was begun a few minutes later and shows the right foot to be rapidly warming, whereas the left foot (post-ganglionic fibres cut) is cooling towards room temperature, and it remains cool for seventy minutes. Great adrenaline sensitization is present; its degree could not be determined owing to the difficulty of re-establishing vasodilatation in the sympathectomized foot.

less sensitive to adrenaline than denervated vessels; nevertheless sensitization is unequivocal. The corresponding operation in the lumbar region yields essentially

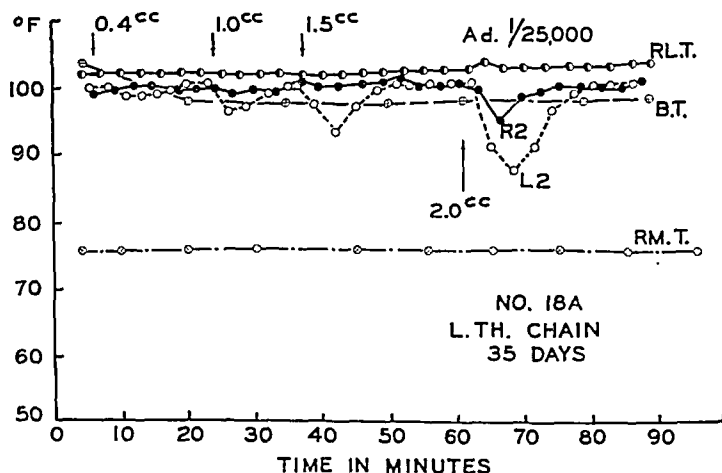


FIG. 565.—Monkey SS 18A, one month after division of the left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show the degree of adrenaline sensitization developing after pre-ganglionic sympathectomy; in this case about two to three times normal. In some animals the sensitization is greater, up to five times normal.

similar results. In our animals adrenaline sensitization developed between twenty-four and forty-eight hours after sympathetic nerve section (*Fig. 566*); it has not increased perceptibly after a week, and from that time it has persisted unchanged for as long as we have observed our animals, which in some cases is eleven

months. The time of onset of sensitization is apparently independent of the distance from the lesion to the end of the severed nerve fibres.*

Sympathectomized vessels become sensitized to other vasoconstrictor substances besides adrenaline (Dale and Richards⁷). We have tested the effects of injections of pitressin (Parke, Davis & Co.—20 pressor units per c.c.) and ephedrine, after pre- and post-ganglionic sympathectomy, and find that the sensitization to these substances differs in one striking feature from that seen with adrenaline. When the post-ganglionic fibres are cut the degree of sensitization is never great, and it may be so

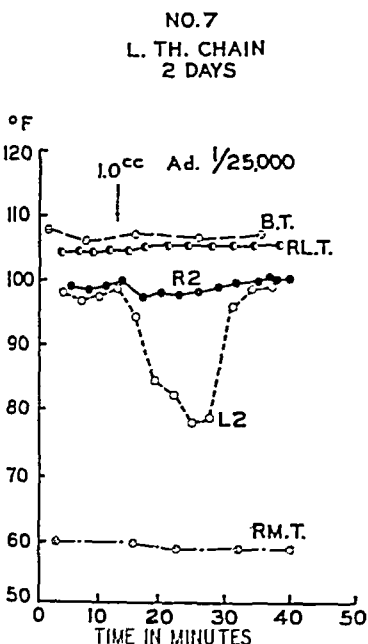


FIG. 566—Monkey SS 7, forty-eight hours after division of left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL T., Rectal temperature; B T., Box temperature; RM. T., Room temperature. To show that adrenaline sensitization develops within forty-eight hours of division of vasomotor fibres. In this animal sensitization is already nearly maximal.

4. **Subsidiary Findings.**—Arising out of the foregoing experiments are three points, not directly concerned with the main thesis, which may be mentioned here:—

a. *Sympathetic Vasodilator Fibres.*—The vasodilatation which is induced in normally innervated limbs by small doses of adrenaline fails to occur when the sympathetic supply is interrupted either proximal or distal to the sympathetic

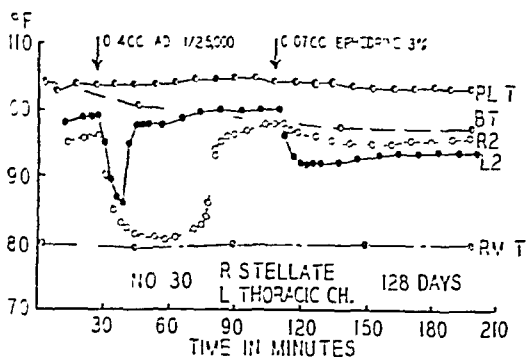


FIG. 567—Monkey SS 30, four months after right cervico-thoracic ganglionectomy, and division of left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL T., Rectal temperature; B T., Box temperature. The right forefinger (post-ganglionic fibres cut) is much more sensitive to adrenaline than the left forefinger (pre-ganglionic fibres cut). In the case of ephedrine the reverse is true.

slight as almost to pass undetected by the skin temperature method. On the other hand, pre-ganglionic lesions render the vessels readily susceptible to both pitressin and ephedrine (Fig. 567).

* It has been suggested that degeneration of the peripheral part of a severed nerve-fibre does not occur concurrently throughout its length, but rather progresses wave-like from the site of section to the periphery. As development of adrenaline sensitization seems to depend on the occurrence of degenerative changes in the severed nerve-fibres, and not simply on interruption of impulses, it seemed possible that the time of onset of sensitization might be proportional to the distance from the section of the fibres to their termination. However, when one ulnar nerve in a monkey was crushed at the wrist, and at the same time the other was crushed in the axilla, neither little finger became sensitized to adrenaline within the first twenty-four hours, and both were sensitized after another twenty-four hours.

ganglia. A small dose of adrenaline is then either without effect or is followed by a fall in temperature (contrast *Fig. 555*), and a rise in temperature has not been seen no matter how small the dose employed. Nor does adrenaline reversal occur after the administration of ergotoxine (*Fig. 568*). This is what would be expected if the sympathetic vasodilator fibres were sectioned along with the constrictors, as appears to be the case. Because these dilator effects of small doses of adrenaline disappear equally when sympathectomy is performed by sectioning the anterior nerve-roots and because they are unaffected by cutting the posterior roots (p. 813),

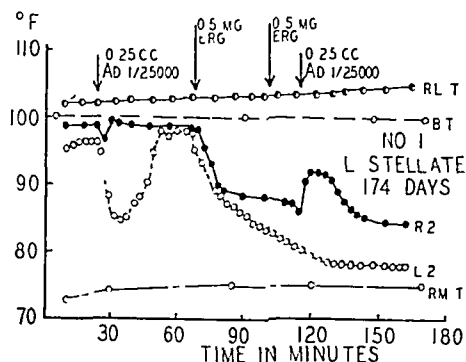


FIG 568—Monkey SS 1, six months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show the phenomenon of adrenaline reversal and its abolition by sympathectomy. When a dose of adrenaline that causes a moderate degree of vasoconstriction is repeated after the injection of ergotoxine, vasodilatation takes place. There is no trace of the reversal phenomenon on the left, sympathectomized, side

it is probable that most or all sympathetic dilator fibres concerned in the adrenaline reaction under discussion leave the spinal cord in the anterior roots, and not in the posterior roots.

b. Recovery of Vasomotor 'Tone' in Arm and Leg.—In order to counteract the effect of gravity, the normal tone of leg vessels exceeds that of arm vessels,* so accounting, it is said, for the superior results obtained by lumbar sympathectomy in man. But the operation practised on the upper and lower limbs differs fundamentally, and this difference, as we have seen, appears to be enough to account for the difference in clinical results in vasospastic cases of equal severity. When the arm and leg are subjected to the same type of sympathectomy (pre- or post-ganglionic),

the effect is strikingly similar in both limbs. There is, however, some difference. Comparison of *Fig. 559* with *Fig. 564* and of *Fig. 561* with *Fig. 562* reveals that recovery of tone in vessels of the leg tends to exceed that in the arm, the opposite of what has been supposed. It seems that when blood-vessels are deprived of their nerve-supply they tend to assume a state of tone approaching the average normal.

c. Sweating and Raynaud's Syndrome.—The third point to which attention may be drawn refers to the periodic attacks of vasospasm which characterize Raynaud's syndrome. It has been shown that blood-vessels deprived of their post-ganglionic sympathetic fibres usually soon begin to respond to cold much as do normal vessels. Yet the results of cervico-thoracic ganglionectomy for Raynaud's syndrome are not so bad as this fact might lead one to suppose. It is widely believed that abnormal vasoconstrictor impulses of central origin operate in many cases of Raynaud's syndrome and that their interruption by sympathectomy is an important factor in removing the disposition to attacks of vasospasm. But it must not be forgotten that sympathetic discharges of this nature involve the whole sympathetic apparatus, including the adrenal medulla. While post-ganglionic sympathectomy removes the possibility of nervous vasoconstriction of central origin, it renders the

* The same argument holds for monkeys, since they spend much of their time semi-erect or climbing.

vessels so sensitive to adrenaline, that release of 'physiological' quantities from the adrenal glands, or release of an adrenaline-like body from a source other than the adrenals (Grant¹³), may exert a profound vasoconstrictor effect which may well exceed that normally resulting from sympathetic nervous impulses alone.

There is another factor to be taken into account which has not received the attention it perhaps merits. A well-known extra-vascular effect of sympathectomy whose influence on the temperature of the skin may be overlooked is the abolition of sweating. Monkey No. 31 often sweated profusely when he became warm, and he serves well to illustrate the point at issue. In this animal the left ulnar nerve was crushed at the wrist. The usual degree of adrenaline sensitization developed in the little finger, being maximal, or nearly so, when the record was taken (Fig. 569). A moderate dose of adrenaline provoked the usual vasoconstrictor response on the sympathectomized side, with a fall of 17° F., and recovery after thirty

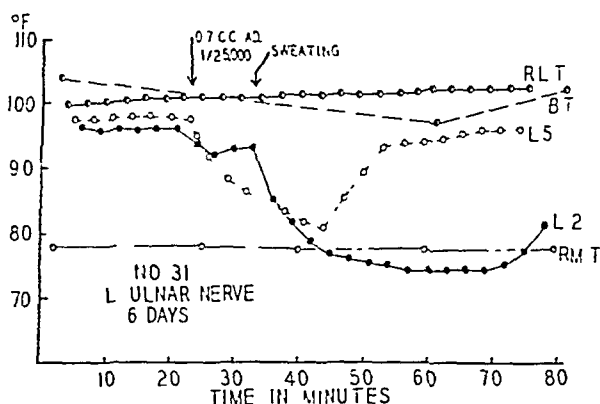


FIG 569—Monkey SS 31, six days after crushing left ulnar nerve at wrist. L2 and L5, Left forefinger and little finger, RLT, Rectal temperature, BT, Box temperature, RMT, Room temperature. To show profound cooling effect of sweating. The normal finger is at first a little cooler than the denervated finger (post-ganglionic fibres cut), probably on account of slight invisible sweating. When adrenaline is injected the usual vasoconstriction due to sensitization occurs in the little finger, lasting thirty minutes. Slight vasoconstriction of the normal finger also takes place, it begins to pass off, then the temperature of the finger falls rapidly and remains below that of the room for thirty minutes. This secondary fall in temperature coincided with the appearance of beads of sweat on the undenervated part of the hand.

minutes. The temperature of the forefinger (normally innervated) of the same hand fell 6° F., and began to warm as the effect of the adrenaline wore off; then it fell again unexpectedly. At this time the whole hand except the little finger and the ulnar side of the ring finger was seen to be damp with sweat. The forefinger continued to cool to a point 3° F. below the room temperature, and only began to warm fifty minutes after the injection, and twenty minutes after the temperature of the sympathectomized adrenaline-sensitive finger had returned to its original level. The victims of Raynaud's syndrome sweat profusely about the hands and feet. Evaporation of sweat cools the skin, and consequently the superficial vessels. These, already narrowed by vasoconstrictor impulses, and perhaps by circulating vasoconstrictor substances, constrict further under the influence of cold, each factor increasing the effect of the others, and a prolonged attack of vasospasm may result. Any form of sympathectomy, by abolishing sweating, may prevent the formation of the vicious circle, and in this way alone may substantially reduce the number of attacks.

D. FACTORS CONCERNED IN THE RECOVERY OF VASOMOTOR ACTIVITY (TONE) AFTER SYMPATHECTOMY

An attempt will be made to analyse the vasomotor condition illustrated by *Figs. 560 and 564*, where the part deprived of its post-ganglionic nerve-supply reacts to cold sooner and to a greater extent than the normal part under similar conditions. In normal circumstances the following possible factors may lead to constriction of the cutaneous blood-vessels of the extremities, with consequent cooling of the skin :—

Vasoconstrictor impulses passing along sympathetic vasoconstrictor nerves, or along extrasympathetic nerves.

Inhibition or interruption of vasodilator impulses passing along sympathetic vasodilator nerves, or along extrasympathetic nerves.

Vasoconstrictor substances formed in the body, three of which are known, namely adrenaline, sympathin, and pituitrin (the first two may be identical).

Lack of normal vasodilator substances, *e.g.*, histamine-like bodies such as H-substance described by Lewis.

Direct effect of cold on vessels.

1. Interruption of Vasomotor Impulses.—

Vasoconstrictor Impulses passing along Sympathetic Nerves.—Impulses of central origin can play no part, for the sympathetic nerves have been cut and post-ganglionic fibres have degenerated. Kuntz¹⁷ states that there are no ganglion cells along the walls of blood-vessels from which impulses might arise; also the presence of ganglion cells favours relative vasodilatation rather than constriction (*Figs. 561, 562*).

Extra-sympathetic Vasoconstrictor Nerves.—It may be stated here that there is no satisfactory evidence for the presence of such nerves. This point is discussed on pages 812 *et seq.*

Interruption of Vasodilator Impulses.—Sympathetic vasodilator nerves accompanying the vasoconstrictor fibres are almost certainly present in many kinds of animals, including monkey and man, and as they accompany the vasoconstrictor fibres, sympathectomy destroys them. It is known that vessels which have been denervated for some time are prone not to relax completely when the body is warmed, the temperature of the part they supply rarely becoming quite as high as in the corresponding normal limb. The work of Lewis and Landis,²² and Lewis and Pickering²³ strongly suggests that failure of maximal dilatation is due to interruption of sympathetic vasodilator fibres. But *Figs. 564, 566, and 570* show that the temperature of the denervated part can rise almost as high as that of the normal limb when conditions favour vasodilatation. This indicates that the absence of vasodilator impulses probably plays little part in the recovery of contractile power of sympathectomized vessels.

Interruption of Extra-sympathetic Vasodilator Impulses.—This does not concern the present issue and is discussed on page 812 *et seq.*

2. Vasoconstrictor Substances.—

Adrenaline.—Vessels deprived of their post-ganglionic sympathetic supply become intensely sensitive to adrenaline (*Figs. 563, 564*), and amounts of adrenaline which can enter the blood-stream when the adrenal glands are subjected to adequate physiological stimulation are sufficient to cause prolonged vasoconstriction (Freeman *et al.*¹⁰). Freeman, by plethysmographic methods (personal communication), has shown that adrenaline produces a similar constrictor effect

when the limb is kept in water at blood heat, so that this effect is not dependent on some degree of cold.

Pituitrin.—Lewis²⁰ (pp. 237-8) has shown that pituitrin probably plays no part in the normal reactions of skin vessels, at all events the arterioles; Grant¹³ finds that removal of the pituitary body does not influence the reactions of sympathectomized vessels; and we have found only a very mild degree of sensitization to pitressin in denervated vessels.

Sympathin.—We have no experimental evidence to offer concerning the possible role of sympathin in the recovery of vascular tone. The quantity entering the blood-stream under the conditions of these experiments is probably very small (Rosenblueth and Schlossberg²⁶).

Lack of Normal Vasodilator Substances.—Grant¹³ discusses the view that continued hyperæmia may reduce the concentration of vasodilator substances in the tissues, and concludes that "we cannot therefore in this way account for the increased responsiveness of the denervated vessels".

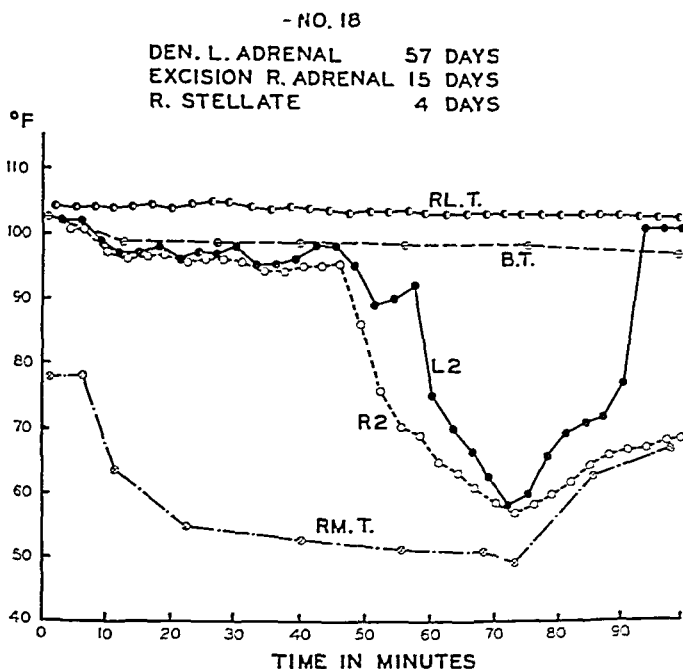


FIG. 570.—Monkey SS 18, two months after denervation of left adrenal, two weeks after removal of right adrenal, four days after right cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers. RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature.

3. *Effect of Cold on the Vessels.*—In two monkeys the adrenal medulla was inactivated by removing one adrenal gland and denervating the other; cervico-thoracic sympathectomy was then performed. Fig. 570 shows that when the hands (and, as usual, the head) are exposed to cold, the sympathectomized fingers cool readily. This record was taken only four days after operation. It was repeated ten days later with a similar result, and the second animal behaved in the same way. Denervated vessels thus constrict when exposed to cold, in the absence of adrenaline.

It is possible that circulating sympathin or pituitrin are partly responsible for vasoconstriction in these circumstances, but the simpler and more acceptable explanation is that the vessels have acquired a direct susceptibility to cold.

Since this was written we have carefully studied Grant's¹³ recent paper on the effects of denervation on the vessels of the rabbit's ear, in which he presents evidence indicating that the recovery of activity in vessels deprived of their sympathetic nerves is largely due to formation of an adrenaline-like body which is not adrenaline, nor pituitrin, and probably is not sympathin. Grant states also that external cold may cause constriction of denervated vessels, in excess of that produced in the normal vessels, but he does not seem to think external temperature is as important a factor in the variations of tone of the denervated vessels of the rabbit's ear as it appears to us to be in the monkey's hands and feet. This subject is pursued in the next section. It is of course recognized that cold may act by liberating an adrenaline-like substance.

4. Combined Effect of Cold and Adrenaline.—Either adrenaline or cold acting alone may exert a very considerable constrictor effect on sympathectomized vessels (*Figs. 563, 564, 570*); combined, the sum of their separate effects is far

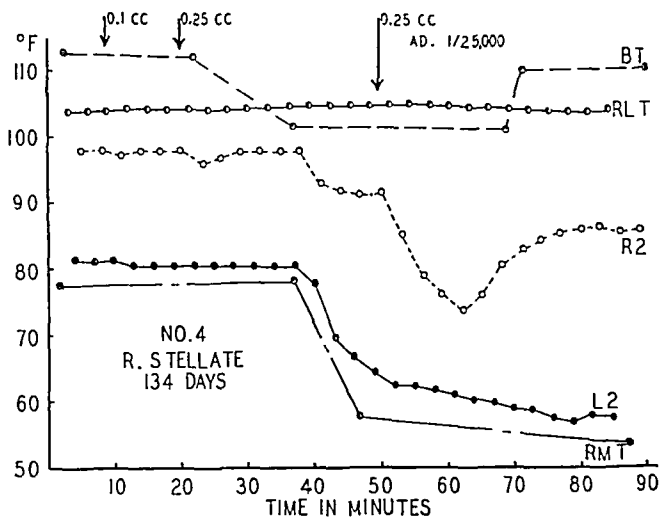


FIG. 571.—Monkey SS 4, four months after right cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show the combined effect of adrenaline and external cold. This is the sole animal that showed a relatively persistent vasodilatation in a limb deprived of its post-ganglionic fibres; half the usual degree of adrenaline sensitization developed. At autopsy the lesion appeared to be complete on naked-eye dissection.

exceeded. *Fig. 571* shows part of a record obtained from the single animal which maintained a considerable degree of vasodilatation after cervico-thoracic sympathectomy, and which also showed less adrenaline sensitization than any other animal with a similar lesion. The chart demonstrates that a part not very sensitive to adrenaline when the room is warm may exhibit much greater sensitization when the room is cold, body temperature remaining well above normal. The fall in temperature of the fingers on cooling the room was in simple proportion to the fall in room temperature, showing the rate of blood-flow to be the same when each injection of adrenaline was made.

When the temperature of the body as a whole is reduced, an over-reaction to adrenaline is still clearer. An animal with a pre-ganglionic lesion was used in this experiment, as it has not been found possible to re-establish vascular dilatation in a part without post-ganglionic fibres under requisite conditions. As shown in Fig. 572, a small dose of adrenaline was followed by a fall in temperature, the rectal temperature being 103° F. The rectal temperature was then reduced to 97.5° , and a tenth of the initial dose of adrenaline produced a fall in temperature through an equal range. Comparison of the temperature ratios shows that the rate of blood-flow was the same when each injection was given. This increase in sensitivity might be thought only apparent, on the grounds that adrenaline or an adrenaline-like substance is produced when the temperature of the body falls.

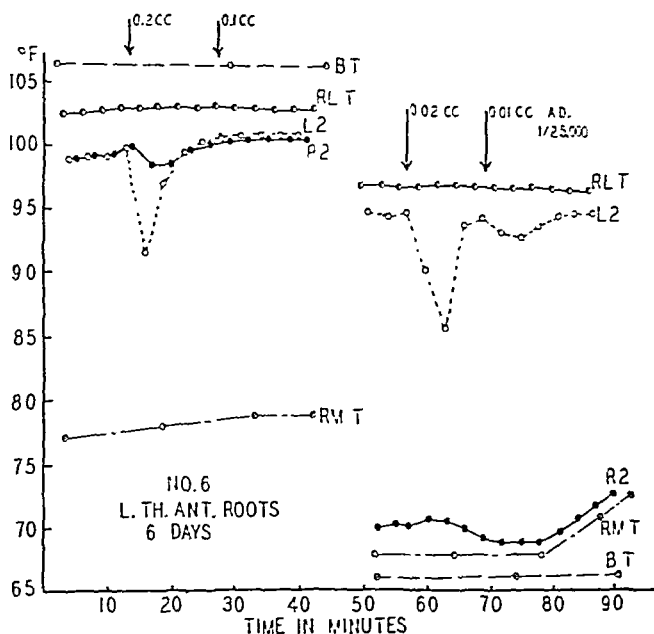


Fig. 572.—Monkey SS 6, six days after intradural division of left thoracic anterior nerve-roots 3 to 9. R2 and L2, Right and left forefingers; R.L.T., Rectal temperature; B.T., Box temperature, R.M.T., Room temperature. To show the combined effect of low blood-temperature and adrenaline.

But full dilatation of the sympathectomized fingers persisted in spite of the low rectal temperature. Since injection of 0.02 c.c. of 1-25,000 adrenaline was followed by considerable vasoconstriction, and since half this quantity produced only a minimal effect, it follows that the amount of effective constrictor substance that may have been formed in the body must be minute (very much less than the equivalent of $\frac{1}{10}$ ths of the first dose of adrenaline injected), and any apparent sensitizing effect produced in this way is therefore probably of minor importance. It is possible, however, that some substance is produced which, having little effective constrictor action itself, may yet serve as a powerful adjuvant to adrenaline.

Grant has shown that his adrenaline-like body is formed under conditions of muscular or nervous activity. In the experiments described in this paper, the monkeys were under the influence of dial. They were sufficiently deep asleep to

fail to react to the stimulus of injections into the temporal muscle except by a fleeting grimace. When rabbits are observed under such conditions, Grant finds that the adrenaline-like substance does not appear to be present in active quantities.

To summarize: the blood-vessels of an extremity deprived of their post-ganglionic nerve-supply soon regain activity. This recovery of contractile power to physiological stimuli often occurs to such a degree that in certain circumstances where the normal limb remains warm, the denervated limb tends to assume the temperature of the surrounding air. Among the more important of the many possible factors responsible for this abnormal cooling reaction is an increased sensitivity of the denervated vessels to the separate or combined effects of cold and adrenaline (or an adrenaline-like substance).

E. THE PHENOMENON OF ADRENALINE SENSITIZATION

It has been known for many years that smooth muscle whose motor nerves are derived from the sympathetic system becomes hypersensitive to adrenaline when these nerves are cut, and this phenomenon as exhibited by the blood-vessels of the rabbit's ear was described by Meltzer and Meltzer²⁴ in 1903. It is to James White that we owe the first recognition of the importance of sensitization to sympathetic surgery. It has been shown that the vasoconstrictor effect of adrenaline on denervated vessels is increased about ten-fold (*Fig. 563*). Sensitization also develops constantly after pre-ganglionic lesions of the sympathetic trunk, but to a less degree—about three times normal (*Fig. 565*). It seemed possible that some or all of the adrenaline sensitization that develops after trunk lesions of this type was due to concomitant division of a few post-ganglionic fibres, in spite of all such lesions being made proximal to the first grey ramus in question, at all events where the arm was concerned (*Fig. 553*). For example, it might be that some fibres to the arm arising from the lower thoracic roots relay in the lower ganglia of the thoracic sympathetic chain, and that post-ganglionic fibres ascend in the chain to reach the second thoracic ganglion from which springs the first grey ramus to the arm. Langley¹⁹ wrote, "Although each ganglion of the lateral chain sends the great majority of its fibres to the corresponding somatic nerve, some ganglia commonly send a few fibres to the nerve above, and some send a few both to the nerve above and the nerve below". To clinch the point, decentralization of the upper limb was carried out in five monkeys by dividing the anterior roots on one side from thoracic segments 3 to 9 inclusive. Adrenaline sensitization developed just as it does when the thoracic chain is cut immediately below the second thoracic ganglion.

Moreover, sensitization of about the same intensity occurs when the vasomotor pathways are interrupted at a higher level, namely, in the spinal cord (*Fig. 573*). On the other hand, if lesions are made above the vasoconstrictor centre in the medulla oblongata, the sensitization phenomenon is considerably reduced.

Some difference in the vasoconstrictor reaction on the two sides of the body is still found, however, when lesions of the vasomotor fibres are made at a higher level than the medulla.³ Here a difficulty arises, for even if we assume that unilateral lesions of the nervous system above the medulla affect the vessels of only one side of the body, it is not easy to decide which is the normal side and which the abnormal, since the changes produced are not great. The left cerebral peduncle has been divided in one animal, causing a complete loss of voluntary movement

of the right arm and leg, and a complete *left* piloparesis. A week later the vasoconstrictor reaction to adrenaline was distinctly greater in the right (paralysed) fingers than the left, the left piloparesis remaining complete. These findings could be explained by assuming that the vasomotor fibres decussate in the lower part of their course as they descend to the vasomotor centres in the medulla, and that the pilomotor fibres in the same region decussate high or not at all. The same difficulty clouds the meaning of vascular changes following lesions above the thalamus. Thus, when the motor and premotor cerebral cortex (areas 4 and 6 of Brodmann) is removed, the consequent relative vasodilatation usually seen on the opposite side of the body is not accompanied by any trace of adrenaline sensitization; in fact vasoconstriction may be less than that on the same side as the lesion. Unilateral cortical lesions cause a consistent difference between the vascular reactions on the two sides of the body, but this difference is small enough to raise doubt as to the side on which the change has occurred.

It does not yet seem possible to draw definite conclusions with regard to vascular effects of injury to the brain at levels higher than the medulla. It seems *legitimate* to state, however, that unilateral adrenaline sensitization will follow division of the vasomotor fibres at any level below the medulla, and probably at any level below the caudal portion of the thalamus; and that the fewer the number of cell stations there are between the lesion and the periphery, the greater will be the degree of sensitization.

There is no reason to doubt that the sensitization developing after denervation is due to change in the peripheral structures. In the case of decentralization one might readily

conclude that the change, whatever it may be, leading to sensitization takes place in the decentralized ganglia of the sympathetic chain. Feldberg and Gaddum⁸ have presented evidence showing that the chemical mediator responsible for the transmission of impulses across the synapses of sympathetic ganglia is not adrenaline but acetylcholine, and we have been able to demonstrate what appears to be acetylcholine sensitization in decentralized ganglia (*Fig. 574*). Cannon and Rosenblueth⁵ (1936) have recently shown the same phenomenon in connection with the superior cervical ganglion and the nictitating membrane, and they appear to have proved that the increased effect of acetylcholine after decentralization is due to true ganglionic sensitization and not to any other cause. Hence it is

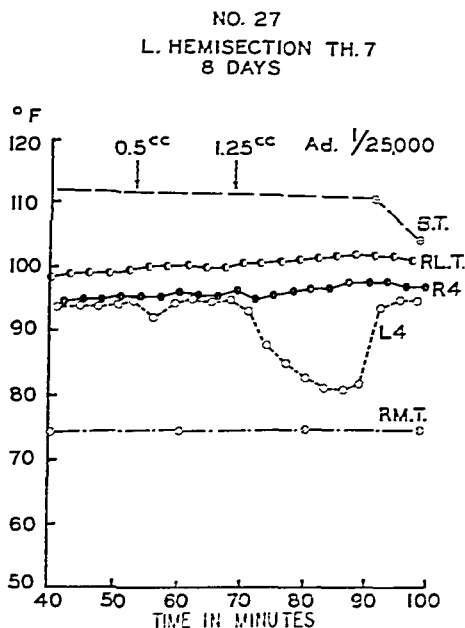


FIG. 573.—Monkey SS 27, eight days after section of left half of spinal cord at seventh thoracic segment. R4 and L4, Right and left fourth toes; R.L.T., Rectal temperature; S.T., Box temperature; R.M.T., Room temperature. To show that definite adrenaline sensitization develops after interruption of the vasomotor fibres at levels as high as the spinal cord.

probable that adrenaline sensitization of decentralized vessels is an expression of some change at or near the vessels themselves. The nature of this change is obscure. Adrenaline is normally quickly destroyed in the body by oxidation. According to Bacq,¹ when substances which retard oxidation are injected into the

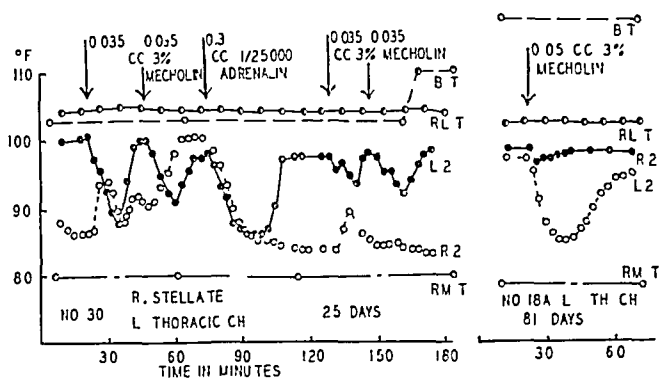


FIG. 574—Monkey SS 30, twenty-five days after right cervico-thoracic ganglionectomy and division of left thoracic chain at third ganglion, and Monkey SS 18A, eleven weeks after division of left thoracic chain at third ganglion R2 and L2, Right and left forefingers, RL T, Rectal temperature; B T, Box temperature, RM T, Room temperature. Intramuscular injection of a dose of acetylcholine of the size used in this experiment is followed by a fall in temperature or no change in the normally innervated fingers. After pre-ganglionic sympathectomy, a considerable fall occurs, always much greater than on the normal side, as shown in the short chart to the right. Even if the normal or pre-ganglionectomized parts are initially cool, a rise in temperature is not seen when acetylcholine is injected. The longer chart on the left shows that such doses of acetylcholine cause dilatation of vessels whose post-ganglionic fibres are cut.

blood-stream the animal is sensitized to adrenaline, and a given dose is followed by an effect several times normal. It may be that sympathectomy induces some change at or near the neuromuscular junction that prolongs the normal rate of oxidation of adrenaline. Rosenblueth²⁷ has suggested that sensitization is a result of increased permeability of the cells to adrenaline.

F. EXTRASYMPATHETIC VASOMOTOR CONTROL

The classical experiments of Bayliss² prove that stimulation of the distal cut end of the posterior roots causes a considerable vasodilatation in the peripheral part concerned. Bayliss showed that the sympathetic system plays no part in this reaction. He ascribed it to antidromic conduction along somatic sensory fibres, and until recently most physiologists have uneasily accepted his explanation. In 1933 Bishop, Heinbecker, and O'Leary⁴ showed that the posterior nerve-roots contain a group of slow-conducting, non-myelinated, motor vasodilator fibres ("C" fibres) which do not enter the sympathetic rami or chains, whose cells of origin are in the posterior root ganglia "accessible to reflex activation via synapses within the cord, and that none of such fibres mediate any of the commonly tested 'afferent functions'". This work has been said by White³³ (p. 66) to render unnecessary the uncomfortable theory of antidromic conduction, and to provide an acceptable mechanical explanation for the observed fact of posterior root vasodilatation under experimental conditions. But the problem of the biological significance, if any there be, of this vasodilator mechanism which can be activated under experimental conditions remains in large part unsolved. Lewis and his associates^{21, 22, 23} discovered, however, that when an area of skin is rapidly supercooled by immersion in ice-water, a brisk vasodilator reaction occurs, limited to the part

so exposed. They showed further that this reaction is not abolished by sympathectomy and degeneration of the vasomotor fibres, nor is it abolished immediately after division of all mixed nerves to the part, but that it disappears if time is allowed for the mixed nerves to degenerate. It is difficult to see how the vasodilatation observed in these experiments can occur when the mixed nerves are cut (but not degenerated) without the aid of antidromic conduction.

No matter how we may interpret the results of the extensive investigations concerning the problem of 'posterior root vasodilatation', we cannot escape the conclusion that there exists a potent peripheral vasodilator mechanism, which is dependent upon the mixed nerves, and which may be susceptible to reflex influence, perhaps involving the central nervous system. We have therefore carried out experiments designed to test the possible bearing of such a mechanism on the treatment of vasospastic states of the extremities.

These experiments suggest that the reactions of the peripheral vessels of non-muscular parts of the limbs to changes in temperature of ordinary degree are not in any important way dependent on the integrity of the posterior root fibres. The special conditions necessary to elicit Lewis's vasodilator reaction have not been used in this work, air being the only cooling agent employed. In three monkeys the left upper limb was de-afferented by dividing the posterior nerve-roots from C.4 to Th.3 between the posterior root ganglia and the spinal cord. In all three animals the operated fingers tended to remain about 3° F. warmer than their fellows for about a month, when the temperatures became equal on both sides. The reactions to variations in external temperature and to adrenaline were otherwise the same as on the normal side. Full vasodilatation can occur, and does so as readily as on the normal side, and a rise in temperature, equal in the two hands, may also follow administration of a small dose of adrenaline. In one of these animals left cervico-thoracic sympathectomy was performed seventy-seven days after de-afferentation. The immediate and late effects were indistinguishable from those seen in animals with intact posterior roots. In one monkey the posterior root ganglia from C.4 to Th.3 were removed. Immediately after operation the affected fingers remained warm in spite of low rectal and room temperature (this dilatation may have been due to coincident irritation or injury to grey rami, but adrenaline sensitization did not develop, showing that the grey rami suffered no lasting damage). The second record from this animal, taken ten days later, and subsequent records, show that the operated fingers assume almost the same temperature as the normal ones, and show no abnormal temperature or adrenaline reactions. Because the posterior root fibres concerned with vasomotor activity appear to be exclusively vasodilator, the reactions of the vessels of de-afferented limbs to acetylcholine have been closely observed. There was never any trace of sensitization to this substance, whether the posterior root ganglia were intact or not. Lewis believes that posterior root vasodilatation occurs by the liberation of histamine or a histamine-like body in the neighbourhood of the vessels, whereon it acts directly, hence the absence of sensitization to acetylcholine after post-root section might have been predicted. It may be added that the vasomotor reaction to histamine, whether injected locally or intramuscularly at a distance, has been modified in no consistent way in our animals by any particular nerve lesion.

In one monkey the anterior nerve-roots supplying the foot were sectioned within the dura on one side (roots L.4 to S.3), thus paralysing the voluntary muscles

of the leg but leaving the sympathetic supply to the foot vessels intact. No modifications in the reactions of the blood-vessels of the toes to temperature changes or to injected adrenaline or acetylcholine were observed.

Section of all the mixed nerves to a part had the same effect on the reactions of the blood-vessels as sympathetic ganglionectomy (post-ganglionic division), as observed by our methods, and this agrees with the findings of Grant, Bland, and Camp.¹⁴

We may therefore conclude that the types of temperature reaction described in this paper are not materially influenced by nerve lesions which do not involve the vasomotor fibres of the sympathetic system.

IV. SUMMARY AND CLINICAL INFERENCES

The early and late effects of division of the vasomotor fibres at various levels have been studied in the Rhesus monkey by means of skin-temperature records.

Twelve monkeys have undergone cervico-thoracic sympathetic ganglionectomy. In only one of these has a good degree of vasodilatation persisted, and in at least two animals the sympathectomized side was more sensitive to cold than the normal side. Eleven of the twelve animals developed a very considerable degree of adrenaline sensitization, about ten times normal, while the remaining monkey, that just mentioned, was about half as sensitive as the others. In two monkeys the lumbosacral sympathetic chain from the renal vein to the lower end of the sacrum has been excised. Both behaved much like the more sensitive cervico-thoracic animals. There are many possible factors to account for recovery of activity (or 'tone') in sympathectomized vessels—enhanced susceptibility to external cold, to adrenaline, and to some adrenaline-like substance produced in the body, are of special importance.

In eight monkeys the thoracic chain has been cut just above or below the third thoracic ganglion. In none of these has the sympathectomized side constricted as rapidly, to a greater extent, or for as long a time as the normal side on exposure to cold. Adrenaline sensitization develops, but it becomes only about one-third as intense as after post-ganglionic division. In two monkeys the lumbar chain has been removed from the renal vein to the level of the bifurcation of the aorta. In both vasodilatation has persisted, and adrenaline sensitization has approximated to that seen after pre-ganglionic division of the vasomotor fibres in the upper limb.

The experimental data show that pre-ganglionic operations are much more effective in producing chronic vasodilatation than post-ganglionic operations, so agreeing with clinical observations. The conventional operation for vasospastic states of the upper limb is not incomplete but too complete, and it would seem rational to replace it by a pre-ganglionic operation whose worth in the lower limb has already been proved clinically.^{34,* 31} In man, the pre-ganglionic fibres to the arm arise from the second to the sixth or seventh thoracic anterior roots (Foerster⁹). There is no need to perform an extensive laminectomy to divide these fibres by direct attack on the anterior roots; all the pre-ganglionic fibres may be

* White states, "This operation has now been performed twenty-eight times on eighteen patients at the Massachusetts General Hospital. Observations over a period of one and one-half years have demonstrated that the lasting increase of blood-flow in the arm after this operation can be as great as in the leg."

interrupted by dividing the thoracic sympathetic chain just below the second thoracic ganglion and by severing the connections to the second thoracic nerve. By this means all the post-ganglionic fibres to the brachial plexus are preserved, and Horner's syndrome is avoided.

The operation usually performed for vasospastic disease of the lower limb has stood the test of time and there is no real reason to modify it. From the theoretical and experimental point of view, it would seem best to leave the fourth lumbar ganglion intact, taking only the second and third, for no white ramus reaches the fourth ganglion and it gives off a grey ramus carrying post-ganglionic vasomotor fibres to the leg.

The feasibility of attacking the vasomotor fibres to both the arm and leg at the same time as they lie in the spinal cord has been considered and rejected, for the arm fibres lie too deep and too close to the pyramidal tract to render the operation practicable (*Fig. 556, B*).

It is with great pleasure that I express my thanks to Professor John Fulton for suggesting the problem of adrenaline sensitization, for the generous facilities afforded me in his laboratories, and for his continued interest and aid.

The early experiments were carried out in collaboration with Dr. Harry Botterrell, now of the General Hospital, Toronto.

REFERENCES.

- ¹ BACQ, Z. M., "Recherches sur la Physiologie et la Pharmacologie du Systeme nerveux autonome. XX. Sensibilisation a l'Adrénaline et a l'Excitation des Nerfs adrén-ergiques par les Antioxygenes." *Arch. internat. de Physiol.*, 1936, xli, 340-366.
- ² BAYLISS, W. M., "On the Origin from the Spinal Cord of the Vasodilator Fibres of the Hind-limb, and on the Nature of these Fibres", *Jour. of Physiol.*, 1901, xxvi, 173-209.
- ³ BEATTIE, J., BROW, G. R., and LONG, C. N. H., "Physiological and Anatomical Evidence for the Existence of Nerve Tracts Connecting the Hypothalamus with Spinal Sympathetic Centres", *Proc. Roy. Soc.*, 1930, cvi, 253-275.
- ⁴ BISHOP, G. H., HEINBECKER, P., and O'LEARY, J. L., "The Function of the Non-myelinated Fibers of the Dorsal Roots", *Amer. Jour. Physiol.*, 1933, cvi, 647-669.
- ⁵ CANNON, W. B., and ROSENBLUETH, A., "The Sensitization of a Sympathetic Ganglion by Pre-ganglionic Denervation", *Ibid.*, 1936, cxvi, 408-413.
- ⁶ DALE, H. H., "On some Physiological Actions of Ergot", *Jour. of Physiol.*, 1906, xxxiv, 163-206.
- ⁷ DALE, H. H., and RICHARDS, A. N., "The Vasodilator Action of Histamine and of some other Substances", *Ibid.*, 1918, li, 110-165.
- ⁸ FELDBERG, W., and GADDUM, J. H., "The Chemical Transmitter at Synapses in Sympathetic Ganglion", *Ibid.*, 1934, lxxxi, 305-319.
- ⁹ FOERSTER, O., "Ueber Störung der Thermoregulation bei Erkrankungen des Gehirns und Rückenmarks und bei Eingriffen am Zentralnervensystem", *Jahrb. f. Psychiat. u. Neurol.*, 1935, lii, 1-14.
- ¹⁰ FREEMAN, N. E., SMITHWICK, R. H., and WHITE, J. C., "Adrenal Secretion in Man. The Reactions of the Blood Vessels of the Human Extremity Sensitized by Sympathectomy, to Adrenalin and to Adrenal Secretion Resulting from Insulin Hypoglycemia", *Amer. Jour. Physiol.*, 1934, cvii, 529-534.
- ¹¹ GASK, G. F., and ROSS, J. P., *The Surgery of the Sympathetic Nervous System*, 1934. Baltimore: William Wood & Co.
- ¹² GASKELL, W. H., *The Involuntary Nervous System*, 1916. London: Longmans, Green & Co.
- ¹³ GRANT, R. T., "Further Observations on the Vessels and Nerves of the Rabbit's Ear, with Special Reference to the Effects of Denervation", *Clinical Sci.*, 1935, ii, 1-33.

- ¹⁴ GRANT, R. T., BLAND, E. F., and CAMP, P. D., "Observations on the Vessels and Nerves of the Rabbit's Ear with Special Reference to the Reaction to Cold", *Heart*, 1931, xvi, 69-101.
- ¹⁵ HARTMAN, C. G., and STRAUSS, W. I., *The Anatomy of the Rhesus Monkey*, 1933. Baltimore: Williams & Wilkins.
- ¹⁶ JELSMA, F., and SPURLING, R. G., "The Role of Sympathetic Nerve Surgery in Vascular Disorders of the Extremities", *Amer. Jour. Surg.*, 1932, xxviii, 76.
- ¹⁷ KUNTZ, A., *The Autonomic Nervous System*, 1934. Philadelphia: Lea & Febiger.
- ¹⁸ LANGLEY, J. N., *The Autonomic Nervous System*, 1921. Cambridge: W. Heffer & Sons Ltd.
- ¹⁹ LANGLEY, J. N., "The Autonomic Nervous System", *Brain*, 1903, xxvi, 1-26.
- ²⁰ LEWIS, T., *The Blood Vessels of the Human Skin and Their Responses*, 1927. London: Shaw & Sons, Ltd.
- ²¹ LEWIS, T., "Observations upon the Reactions of the Vessels of the Human Skin to Cold", *Heart*, 1929, xv, 177-208.
- ²² LEWIS, T., and LANDIS, E. M., "Some Physiological Effects of Sympathetic Ganglionectomy in the Human Being and its Effect in a Case of Raynaud's Disease" *Ibid.*, 151-176.
- ²³ LEWIS, T., and PICKERING, G., "Vasodilatation in the Limbs in Response to Warming the Body; with Evidence for Sympathetic Vasodilator Nerves in Man", *Ibid.*, 1931, xvi, 33-51.
- ²⁴ MELTZER, S. J., and MELTZER, C., "On the Effects of Subcutaneous Injection of the Extract of the Suprarenal Capsule upon the Blood Vessels of the Rabbit's Ear", *Amer. Jour. Physiol.*, 1903, ix, 252-261.
- ²⁵ ROSENBLUETH, A., and CANNON, W. B., "Studies on Conditions of Activity in Endocrine Organs. XXVIII. Some Effects of Sympathin on the Nictitating Membrane", *Ibid.*, 1932, xcix, 398-407.
- ²⁶ ROSENBLUETH, A., and SCHLOSSBERG, T., "The Sensitization of Vascular Responses to 'Sympathin' by Cocaine and the Quantitation of Sympathin in Terms of Adrenalin", *Ibid.*, 1931, xcvii, 365-374.
- ²⁷ ROSENBLUETH, A., and CANNON, W. B., "The Adequacy of the Chemical Theory of Smooth Muscle Excitation", *Ibid.*, 1936, cxvi, 414-429.
- ²⁸ ROSS, J. P., "The Results of Sympathectomy" *Brit. Jour. Surg.*, 1935, xxiii, 433-443.
- ²⁹ SIMPSON, S. L., BROWN, G. E., and ADSON, A. W., "Raynaud's Disease. Evidence that it is a Type of Vasomotor Neurosis", *Arch. of Neurol. and Psychiat.*, 1931, xxvi, 687-718.
- ³⁰ SMITHWICK, R. H., FREEMAN, N. E., and WHITE, J. C., "Effect of Epinephrine on the Sympathectomized Human Extremity. An Additional Cause of Failure of Operations for Raynaud's Disease", *Arch. of Surg.*, 1934, xxix, 759.
- ³¹ TELFORD, E. D., "Sympathectomy. A Review of One Hundred Operations", *Lancet*, 1934, i, 226; 444-446.
- ³² TELFORD, E. D., "The Technique of Sympathectomy", *Brit. Jour. Surg.*, 1935, xxiii, 448.
- ³³ WHITE, J. C., *The Autonomic Nervous System*, 1935. New York: The Macmillan Co.
- ³⁴ WHITE, J. C., "Surgery of the Sympathetic Nervous System", *Jour. Amer. Med. Assoc.*, 1936, cvii, 350-353.
- ³⁵ WOOLLARD, H. H., "The Peripheral Sympathetic Nervous System", *Brit. Jour. Surg.*, 1935, xxiii, 425-432.

SHORT NOTES OF RARE OR OBSCURE CASES

TWO UNCOMMON FORMS OF INTESTINAL OBSTRUCTION OCCURRING CONSECUTIVELY: COMPOUND VOLVULUS AND RETROPOSITION OF THE TRANSVERSE COLON

By E. S. J. KING

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THE occurrence, in one patient, of two attacks of intestinal obstruction, each apparently independent of the other, and both due to unusual conditions, seems of sufficient interest to be worthy of record.

HISTORY.—The patient, a male, aged 38 years, was admitted to hospital, having had acute generalized abdominal pain of a colicky type for thirty-six hours. He had suffered from mild attacks of similar pain for twenty-eight years, but these had always previously passed off in a few hours. On this occasion the pain had persisted and become very severe. He had vomited a number of times, and several enemata administered before his arrival at hospital had produced no satisfactory result. He stated that he was certain that he had passed no flatus.

ON ADMISSION.—There was marked abdominal distension and the superficial abdominal veins were more than usually prominent. There was no localized tenderness.

FIRST OPERATION.—Under spinal anæsthesia, a lower right paramedian incision was made. The distension of the small intestine was so marked that observation was difficult. One coil of ileum was more distended than the remainder, though the small bowel below this was not completely collapsed. Manipulation of the distended loop of ileum resulted in the sigmoid colon, which had been incarcerated in the pelvis, being drawn into the wound. This was also tremendously distended. No further progress could be made until the two main loops were deflated, tubes being fixed into them by purse-string sutures. Only gas escaped from them. It was now easy to see that the mesentery of the distended loop of ileum was adherent to that of the sigmoid colon and that both had twisted in a clockwise direction (one complete turn) (*Fig. 575*). The twist was undone and the adhesion divided. As the condition of the patient was not good, the wound was sutured, the two tubes being brought out through the wound.

Progress for the next week was very satisfactory. At the end of this time the two tubes came out. The next day the abdomen became distended again and this was associated with some pain and vomiting. Enemata gave a good result. Turpentine stupes were applied, and though his condition gave rise to some concern

he gradually improved and by the end of the next week he was progressing favourably. It was thought that the condition was due to adhesions following the laparotomy.

He was discharged from hospital one month after admission

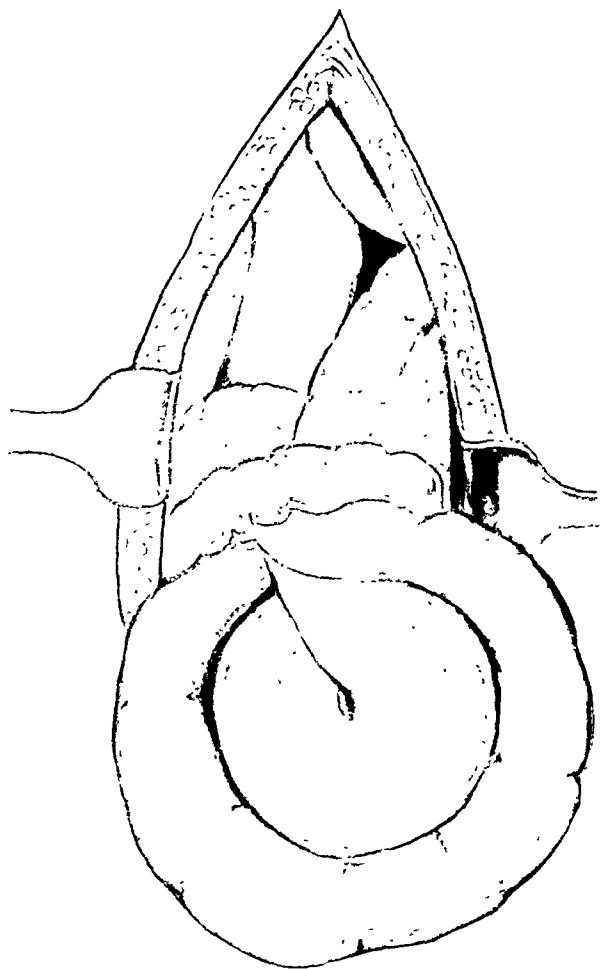


FIG. 575.—Drawing showing the appearance of the intestine at the first operation. There is a complete clockwise twist of the sigmoid colon and a loop of the small bowel.

ON RE-ADMISSION.—He was re-admitted to hospital almost exactly six months after discharge. He had had no further trouble until four days previously, when there had been another attack of generalized abdominal pain. He vomited several times each day and for the thirty hours prior to admission enemata had produced no result whatever. On examination there was extreme abdominal distension sufficient to cause some embarrassment of respiration. It was decided that he had a recurrence of the sigmoid volvulus.

SECOND OPERATION.—Under spinal anæsthesia, incision was made through the scar of the previous operation. The peritoneum was clear except for a few adhesions which were easily divided. No bowel was adherent to the anterior abdominal wall. The sigmoid colon was not distended, though the cæcum and small intestine were markedly so. The ascending colon was followed up into the transverse colon, which was observed to pass behind the superior mesenteric artery (*Fig. 576*). The hiatus behind the mesentery of the small intestine was large enough to accommodate the hand, and the transverse colon could be followed for a short distance towards

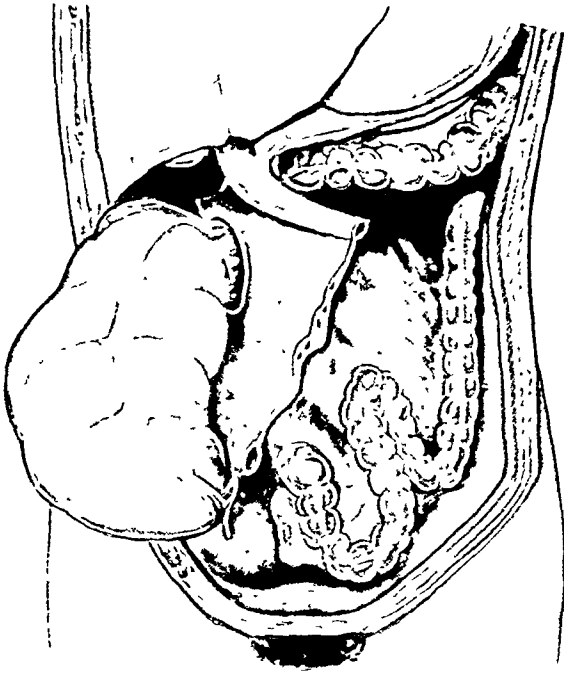


FIG. 576.—Drawing showing the appearance of the ascending colon and transverse colon at the second operation. The transverse colon passed through a large opening in the mesentery of the small intestine. (*Modified from Dott.*)

the left side. A cæcostomy was performed and the wound closed. The patient made an uninterrupted recovery, the distension subsiding rapidly.

Although drainage from the cæcostomy gradually diminished, it did not cease. A barium enema was given and a large sigmoid colon was demonstrated, but no obstruction of the transverse colon was observed and no posterior displacement could be demonstrated with certainty.

THIRD OPERATION.—On Jan. 14, 1936, the abdominal wound was opened. There were a large number of adhesions which could be easily separated. The transverse colon was demonstrated to pass behind the mesentery of the small intestine. The sigmoid colon, though collapsed, was very long. An anastomosis

was made between the cæcum and sigmoid colon. The excess portion of the colon was then brought out through the wound and sutured to the abdominal wall in the manner employed in a Mickulicz operation for carcinoma, and the wound was sutured. On Jan. 19 the protruding loop was excised, and during the next few days the spur was crushed.

On Feb. 10 the cæcostomy was closed, local infiltration anæsthesia being used. The amount of fæcal discharge from the colostomy was then considerably diminished and an easy passage from the proximal to distal loops of sigmoid colon could be demonstrated.

Under local anæsthesia on Feb. 24 the colostomy was closed and the patient made an interrupted recovery.

At the end of January the patient had developed a partial right pneumothorax, which was shown by X-ray examination of the chest and examination of the sputum to be associated with a small tuberculous lesion of the apex of the right lung.

When the wounds were healed he was sent to the country to convalesce.

He was seen again in June, 1936, when he was in an excellent state of health. He had had no further difficulty with his bowels nor abdominal pain.

DISCUSSION

As stated, the interest of this case lies in two unusual forms of obstruction occurring in one patient. Though both are uncommon, neither condition is extremely rare.

In the literature, a compound volvulus involving a loop of small intestine and the sigmoid colon has been described a number of times. Leichtenstein recorded 21 cases, and other cases have been described by Waterhouse and Elfving. The literature, however, has not been searched at all thoroughly.

Very few cases of retroposition of the colon have been recorded in the literature (*see Bibliography*), but this probably is not any index of its frequency, for I have encountered two cases, in addition to the one recorded above, in which this anomaly has been the cause of a subacute intestinal obstruction. In both of these patients—adults in the fourth decade, one male and one female—there was a typical history of attacks of intestinal colic over a period of twenty years and finally an attack of intestinal obstruction sufficiently severe to warrant laparotomy. In both cases anastomosis between the cæcum and sigmoid colon completely relieved the symptoms. Incidentally X-ray examination gave no clue to the presence of the condition, even when the abnormality was known to be present before the examination was made. This has been remarked upon already by other writers.

BIBLIOGRAPHY

Compound Volvulus.—

ELFVING, A. R., *Acta chir. Scand.*, 1925, lix, 185.

LEICHTENSTEIN, quoted by BARNARD, H. L., *Contributions to Abdominal Surgery*, 1910. London.

WATERHOUSE, H. F., *Brit. Med. Jour.*, 1909, i, 1277.

Retroposition of Colon.—

BASTIANELLI, P., *Policlinico*, 1904, xi (Sez. chir.), 56.

BLACK, C. E., *Ann. of Surg.*, 1912, lvi, 888.

BONCI-CASUCCINI, F., *Thèse Lausanne*, 1914.

BORCHGREVINCK, J., *Norsk. Mag. f. Læge.*, 1916, lxxvii, 643.

- BOVERA, A., *Ann. Fac. de med. de São Paulo*, 1927, ii, 21.
 DOTT, N. M., *Brit. Jour. Surg.*, 1923, xi, 251.
 EKEHORN, G., *Arch. f. klin. Chir.*, 1904, lxxii, 572.
 HAUSMANN, F., *Zentralb. f. Chir.*, 1900, xxvii, 19.
 HUNTER, J. I., *Brit. Med. Jour.*, 1922, ii, 800.
 STREHL, H., *Arch. f. klin. Chir.*, 1908, lxxxvii, 8.
 TRUESDALE, P. E., *Jour. Amer. Med. Assoc.*, 1935, civ, 1897.
 TSCHERING, quoted by Ekehorn.
 VIGI, F., *Endocrinol. e pat. costit.*, 1927, ii, 97.

RETROPOSITION OF THE TRANSVERSE COLON

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MRS. E. M., aged 46 years, was admitted under the writer's care to St. Vincent's Hospital, Melbourne, bearing a letter from her family physician, Dr. Grogan, giving the following history of her condition:—

HISTORY.—Laparotomy for (?) intestinal obstruction done fifteen years previously. Of this operation there were no details to be had.

She complained of attacks of abdominal pain which had commenced twelve months previously in the right iliac fossa. These gradually became worse, and her abdomen became distended. A laparotomy was performed, and several large bands of adhesions across the cæcum were cut through. The small intestine and transverse colon were greatly distended, and a mass could be felt to the left of the umbilicus. A cæcostomy was performed. Patient had a stormy convalescence, and returned home with her cæcostomy functioning well. A few days later the cæcostomy closed, and her bowels acted in normal fashion. She began to have further symptoms and signs of intestinal obstruction and a second laparotomy was performed. The original mass was found to have disappeared, but the small intestine and colon were matted together in coils; these were freed as well as possible, and the abdomen closed. Her convalescence was again stormy, but she ultimately recovered, and she returned home very well.

ON ADMISSION.—She stated that for the previous six months she had been having periodical attacks of abdominal pain and vomiting, and distension. Her cæcostomy would then break down, and her symptoms would be relieved. These attacks would last twenty-four hours, and were becoming more frequent.

ON EXAMINATION.—The patient was a rather pale, middle-aged woman. The tongue was coated, but moist. The heart and lungs were clinically clear. On the abdomen were three scars of previous operations. There was a hernia at the site of the cæcostomy wound—no tenderness, no mass palpable, not distended at the time. An enema was given with poor result. On Oct. 19 an enema was given with good result.

OPERATION.—On Oct. 21 the abdomen was opened through a right paramedian lower abdominal incision. Numerous adhesions were divided. The cæcum and ascending colon were found to be enormously enlarged and hypertrophied, and on tracing it up the colon disappeared through an opening in the mesentery of the small intestine. The colon appeared to be kinked or twisted just at this opening; beyond it the transverse colon was normal in size.

On examining the state of affairs, it was found that there was no obvious way of reducing this kink or twist. The only way out was to divide the mesentery, from the hole through which the transverse colon disappeared down to near the terminal ileum, and then to divide the ileum and resect the cæcum and ascending colon up to and through the orifice. This was done, and an end-to-side anastomosis made between the divided ileum and the transverse colon, distal to the orifice. The operation was completed by making a safety-valve ileostomy proximal to the anastomosis and bringing it out through a stab wound. The cæcal bed was drained by a stab wound in the loin. Continuous intravenous saline was given for forty-eight hours after operation.

Her condition improved rapidly and she was discharged on Nov. 7. She has remained well since.

REVIEWS AND NOTICES OF BOOKS

Textbook of General Surgery. By WARREN H. COLE, M.D., F.A.C.S., Professor of Surgery, University of Illinois College of Medicine, and ROBERT ELMAN, M.D., Associate Professor of Surgery, Washington University School of Medicine. Large 8vo. Pp. 1031 + xvi, with 559 illustrations. 1936. London: D. Appleton-Century Co. Inc. 40s. net.

THIS work is the outcome of a course of lectures which has for some years been given to junior students in Washington University. The authors must be congratulated upon writing a comprehensive text-book of surgery in one volume of such a convenient size. The paper upon which it is written and the printing type are excellent. All the illustrations are in black and white, which to the modern student seems rather dull. The photographs are well reproduced, but the diagrams are uninteresting.

There are many innovations which mark this work as no ordinary text-book. For instance, the chapter on "Prophylaxis of Infection—Asepsis and Antisepsis" is full of practical information on operating theatre technique which is not given in the everyday text-book on surgery. The appendix, "Surgical History and Physical Examination", is a valuable addition, but appears to have been an after-thought. Perhaps in the next edition it will become Chapter I. The remark, "Yes, it is surprising how often a careful and expert history will be all that is necessary to lead to a correct diagnosis", will shock the careful surgeon, who bases his diagnosis upon physical signs. The traditional triplet 'Inspection, Palpation, and Percussion', is replaced by "Inspection, Voluntary Movements, and Palpation"; this is a most interesting suggestion.

The chapters on fractures and the genito-urinary system are disappointing. Some of the authors' methods of treatment appear to us to be unorthodox. The plaster cast is not mentioned in the treatment of Colles's fracture. The method of choice in the treatment of keloid, or, again, of cystic hygroma of the neck in an infant—excision—seems untenable. N.A.B. is not mentioned in the treatment of anthrax. Continuous intravenous saline drip infusion is not suggested in the treatment of shock. Neither hypertonic saline nor magnesium sulphate is discussed in the treatment of septic wounds.

The chapter called "Acute Hand Infections" is most practical, and the diagrams illustrating it are instructive. This subject is of the greatest importance to all students, and is often neglected in modern text-books. The fissure of Sylvius surely can never be spelt "Silvius" even in the American language! "Coller" and "Collar" fractures must obviously be misprints, which are not infrequent in other parts of the index. To say that tuberculous ulcers of the tongue may be associated with lupus, but never to mention phthisis, would dangerously mislead the trustful student. That glomus tumours should be classified under neoplasms of nervous origin, rather than as blood-vessel tumours, seems surprising. The suggestion that a routine Wassermann should be performed upon "all private and ward patients" to us would seem grotesque. The chapter on "Surgical Diseases of the Chest", written by Dr. Graham, seems to be the main feature of this book.

The short bibliography, placed at the end of each chapter, will be most useful to the post-graduate student who wishes to read recent original papers and monographs upon the subjects in which he is especially interested.

The importance of a new text-book of surgery depends upon whether it is of greater practical value to the student than the pre-existing and traditional works. This book by Dr. Cole and Dr. Elman will not replace the older books, but it will be a useful work of reference for the teacher of surgery who wishes to broaden his outlook.

The Operations of Surgery. By R. P. ROWLANDS, M.S., F.R.C.S., Late Surgeon to Guy's Hospital; and PHILIP TURNER, B.Sc., M.S., F.R.C.S., Consulting Surgeon to Guy's Hospital. Eighth edition. Vol. II, The Abdomen. Royal 8vo. Pp. 998 + ix, with 514 illustrations (4 in colour). 1937. London: J. & A. Churchill. 36s. net.

THE distinguishing feature about the eighth edition of this well-known book is the fact that the collaboration of other authors has been deemed necessary. Thus Mr. W. H. Ogilvie has

revised and re-written the subject matter on operations for hernia, and on the spleen, pancreas, and rectum; Mr. Ralph Thompson accounts for most of the genito-urinary surgery; Mr. G. F. Gibberd for operations upon the female genital organs; and Mr. R. C. Brock for thoracic surgery. The book necessarily loses some of its traditional character by this arrangement, but the new authors have done their work so well that it still remains one of the best guides for the junior surgeon beginning his operative work. There is indeed a great deal of information on rare and uncommon emergencies and operations, such as the treatment of a strangulated obturator hernia, which bears the stamp of practical experience. It is in this direction that 'Rowlands and Turner' has always excelled in the past, and in a manner which has long remained almost unique.

There are criticisms which could be made. It is regrettable that surgical methods are retained which experience has shown to be obsolete and in fact never indicated. An example of this is the right-sided colopexy operation for constipation. On the other hand, it is quite arguable that surgical values are corrected and rendered more true by a discussion of colectomy for constipation, though for practical purposes this never enters the modern surgeon's mind. It is interesting to read the excellent discussion on the advisability of removing the inflamed appendix when there is an abscess, and also to realize that the argument is based upon what Sir Frederick Treves and Sir Alfred Pearce Gould said in 1905.

There are one or two small points in the urinary section upon which the novice surgeon would be grateful for more detailed information, such as how to deal with the pedicle in sub-capsular nephrectomy and how to find a small stone fixed deep in the kidney. There is far more to praise than criticize in the book, which remains, as formerly, unique in surgical literature.

There seems to be a misprint in the legend to *Fig. 209*.

Modern Urology in Original Contributions by American Authors. Edited by HUGH CABOT, M.D., LL.D., C.M.G., F.A.C.S., Professor of Surgery, The Mayo Foundation, Graduate School of the University of Minnesota; etc. Third edition, thoroughly revised. Royal 8vo. In two volumes. Vol. I, pp. 951, with 546 illustrations and 12 plates; Vol. II, pp. 862, with 374 illustrations and 9 plates. 1936. London: Henry Kimpton. 90s. net.

THE first edition of this book was reviewed in this journal in 1918: it was then stated that the work, as a whole, was "a notable one and creditable to the remarkable body of men who have produced it, and to the Editor, of ancient surgical lineage, who contributed to its articles and moulded and guided its development and birth". This third edition seems to us to be an improvement on the original; it is better bound, the printing is clearer, and the many illustrations are usually excellent—indeed, some of them are really delightful, especially those portraying the anatomy of the genito-urinary tract.

The text has been largely rewritten and brought up to date; there are very few misprints; and, if we must be ungrateful, we think the chief defect is the rather extensive overlapping. We acknowledge the difficulty in avoiding this when there are contributions by so many authors, but there are two distinct articles on polycystic kidney, and the technique of nephrectomy is described several times; they are, however, beautifully illustrated.

On the important question of the estimation of renal function there are sections by both Dr. Braasch and Dr. Elmore Belt; it would be churlish to complain of this, for both discussions are valuable. Many urologists will heave a sigh of relief to find that the authors have had the courage to drop overboard many of the older tests: Ambard's constant no longer appears, even in the index; and cryoscopy and the electrical conductivity of the urine have died and been decently interred—they were probably nothing more than costly and troublesome methods of taking the specific gravity. Of excretory urography Braasch makes the assertion that "any renal pelvis clearly visualized at the end of five minutes must be normal in function". If this generalization is verified in the future, and there seems no reason to doubt that it will be, it will mark an advance of vital importance in kidney surgery and will greatly simplify the task of the surgeon. He is careful to add that the converse does not hold good—"this does not necessarily mean that because a renal pelvis is not well visualized the function is abnormal". It is obvious that there are many factors which may interfere with this. Dr. Elmore Belt gives a full and clearly expressed account of the urea clearance test; he evidently believes in its value, but thinks the necessity of employing a trained chemist its

outstanding drawback. Both authors attach much importance to the use of phenosulphone-phthalein, but recognize that it has limitations in its range of usefulness.

No less than three authors deal with the subject of prostatic obstruction. Dr. Hugh Young writes the article on the pathology and symptoms, and the operation of perineal prostatectomy; Dr. Hermon Bumpus that on transurethral resection; and Dr. Bentley Squier describes the suprapubic operation. The names of these three distinguished surgeons are a guarantee that both text and illustrations are above the average, but we think that Dr. Bentley Squier might have accorded a more generous recognition of Harris's work on the open operation—the latter is very inadequately described.

Dr. Hinman is such an original worker that we expect any section by him to show unusual interest, and, indeed, his article on tumours of the testicle is admirable; in it he discusses the presence of hormones in the urine of such cases and their value in diagnosis, which has been disappointing up to the present. His account of the treatment by radiation and the radical operation is the best we have read, and his results are surprisingly good.

The second volume ends with an additional section on radiation therapy by Dr. Benjamin Barringer; it will be of interest to surgeons on this side of the Atlantic, for he states that his practice is to employ radium exclusively in the form of radon seeds; he thinks that only 10 to 17 per cent of bladder and prostate tumours are radio-sensitive, and that orchidectomy for tumour of the testis should be done only for a more precise diagnosis of the pathology.

It is hardly possible within reasonable limits of space to give an adequate review of such a monumental work, but our general impression is that no better compendium of urology has been published; we feel sure that it will rank as one of the standard works on this subject, and we congratulate Dr. Hugh Cabot and his coadjutors on their achievement.

Kidney Pain: Its Causation and Treatment. By J. LEON JONA, D.Sc., M.D., M.S., F.R.A.C.S., Hon. Assistant Gynaecological Surgeon, Women's Hospital, Melbourne. Large post 8vo. Pp. 94 + viii, with 61 illustrations. 1937. London: J. & A. Churchill Ltd. 7s. 6d.

IN this monograph the author sets out the results of his attempts to apply the methods of the physiologist in the investigation of the kidney pelvis and the ureter. He has made use of both human and animal patients. After catheterizing the ureter, he has injected fluids opaque to the X-rays and observed their behaviour by means of the screen—*pyeloscopy*—and, after connecting the catheter with a manometer, the changes in the intrapelvic pressure—*pyelometry*. Whilst watching the changes that occur in the normal kidney he has come to the conclusion, though his reasoning is not easy to follow, that the renal pelvis never empties itself completely; the residual fluid, which he names "maintenance filling", he estimates at about 8 c.c., and thinks it is dependent on the tone of the pelvis musculature and of that of the ureter. This observation, if confirmed, will almost certainly have an important bearing on the etiology of hydronephrosis. He has tried to find out the effects on the intrapelvic pressure of change of posture, of massaging the kidney, and of the injection of various drugs; of these, the most striking is the effect of sitting the patient up—this produces an astonishing rise of pressure.

A large part of the book is devoted to what he calls "dysfunction", mostly instances of spasm and atony; of this he describes a large variety, and he has studied the action of the injection of drugs on these. He also gives instances of clinical cases, but they are too inadequately described to be entirely convincing.

We think this is a useful monograph, and that it will act as a stimulus to readers who are interested in urology, though they may not agree with the author's generalizations.

Paget's Disease of the Nipple. By KEITH INGLIS, M.D., Ch.M. (Sydney), Professor of Pathology in the University of Sydney; formerly Director of the Kanematsu Memorial Institute of Pathology, Sydney Hospital. Crown 4to. Pp. 233 + xii, with 237 illustrations. 1936. London: Oxford University Press. 36s. net.

THIS monograph gives a detailed study of Paget's disease of the nipple and an interesting survey of the conflicting views that have been put forward from time to time to explain the

nature of the condition. The author supports the view that Paget's disease is a malignant condition affecting the larger ducts of the nipple, and that the malignant cells spread both along the duct epithelium and into the contiguous epidermis of the nipple. The disease tends to run a chronic course, and its extension remains intra-epidermal until a late stage, when the cells break through the basement membrane at some point and give rise to a frank carcinoma.

The appearances in Paget's disease of the nipple are compared and contrasted with those seen in secondary involvement of the nipple in breast carcinoma, duct changes in the breast, extramammary Paget's disease, dyskeratosis, Bowen's dermatosis, and pre-cancerous conditions of the skin generally. Primary tumours of the nipple are also described. The author finds one fundamental difference between precancerous conditions of the skin and true Paget's disease: in the former, the malignant cells arise in the lesion itself; whereas in the latter they arise in a breast duct, and are therefore secondary invaders of the skin.

The book is profusely illustrated and contains numerous admirable photomicrographs, but the subject would seem scarcely worthy of such elaboration.

Terapéutica Quirúrgica. By Dr. JOSÉ ARCE, Professor Titular in the University of Buenos Aires; Director of the Institute of Clinical Surgery, etc. Book IV, Fasc. 1. 10½ × 7 in. Pp 271 + ix, with 321 illustrations. 1936. Buenos Aires: El Ateneo.

The printing and illustrations of this important book are well up to the high standard we have been taught to expect from Buenos Aires.

It is obvious that cerebral surgery is dear to the heart of the Professor of Surgery in the University. He considers that though the mortality of cerebral operations, taken altogether, is high, yet purely operative risks are no worse than in other regions, provided, and *only* provided, they are in the hands of those specially trained for the work. He puts this partly on the ground of the delicacy of neural tissues and of their blood-vessels, but even more, of course, on the necessity for so perfect a neurological investigation as to permit precise visualization of the lesion to be reached.

Moreover, he is a firm advocate of ventriculography with lipiodol or lipiodol and air. For the performance of this method he gives precise instructions, and reproduces a number of very good radiograms. The Professor regards ventriculography as entirely innocuous, and says that no operation in which it can offer any aid should be done without an injection.

After a chapter on generalities, the author deals with methods of approach in one chapter, and then with actual manœuvres on the neural tissues in another. One chapter is devoted to 'classical decompression'; another to the treatment of intracranial hypertension. The next section deals with cerebral injuries. He then speaks of exploratory trephining.

Professor Arce gives an anatomical classification of tumours: those of vessels; of neuroglia; of the 'sympathetic'; of nerves; of the meninges; and of the hypophysis. A final chapter deals with pseudo-tumours. The operative mortality, which includes deaths within two months, is put at round about 20 per cent. The total mortality of glioblastomas is 65 per cent, whilst for pituitary tumours the figure is 25 per cent. These are deaths, at any time, from the disease. We have observed no mention of irradiation in the treatment of cerebral tumours, even as an adjuvant method or for prophylaxis.

The bibliography is extensive, but confined to the literature of South America.

Essentials of Oral Surgery. By Professor V. P. BLAIR, A.M., M.D., F.A.C.S., Washington University, St. Louis, and Professor R. H. IVY, M.D., D.D.S., F.A.C.S., University of Pennsylvania, Philadelphia, with the collaboration of Associate Professor J. B. BROWN, M.D., F.A.C.S., Washington University, St. Louis. Second edition. 9½ × 6 in. Pp. 606, with 445 illustrations. 1936. London: Henry Kimpton. 30s. net.

WRITTEN primarily as a text-book for dental students, this book deals very fully with all practical details of treatment required in oral and dental surgery. It covers the whole field very thoroughly and is well printed and illustrated. Though, as a major surgical procedure, it is outside the range of the book, surgeons may well take exception to the brief reference to the treatment of carcinoma of the tongue and mouth, where the authors fail to do justice to the established value of radium in this condition.

A Manual of Radiological Diagnosis for Students and General Practitioners. By IVAN C. C. TCHAPEROFF, M.A., M.D., D.M.R.E. (Camb.), Assistant Radiologist and Radium Registrar, St. Thomas's Hospital, London. With a Foreword by PHILIP H. MITCHENER, M.D., M.S., F.R.C.S., Surgeon to St. Thomas's Hospital. 9 $\frac{1}{2}$ \times 7 $\frac{1}{2}$ in. Pp. 256, with 286 illustrations. 1937. Cambridge: W. Heffer & Sons Ltd. 21s. net.

THIS work has two great merits: it is short and concise; and the illustrations are well chosen and beautifully reproduced. Mr. Mitchener, who writes a brief commendatory Foreword, stresses the importance of correlating clinical evidence with X-ray findings in making a diagnosis. Each region of the body has first the normal X-ray appearance demonstrated and then those of injury and disease. One hundred and fifty pages are devoted to the bones and joints. In the section on fractures, abnormal conditions, e.g. myositis, are chiefly noted, and space is not wasted in showing typical fractures which present no special diagnostic problem. A very good presentation is made of typical bone tumours: innocent; malignant—primary and secondary. There are 45 pages devoted to the chest, 32 to the gastro-intestinal tract, 14 to the gall-bladder and kidneys; and there are short sections on the female generative organs, tumours of the spinal cord, and on ventriculography.

Medizinische Praxis. Band XXI. Anleitung zur Schmerzbetäubung. By Prof. Dr. FRITZ F. HARTEL, Direktor der chirurgischen Abteilung des Oskar Ziethen-Krankenhauses Berlin-Lichtenberg. Large 8vo. Pp. 106 + x, with 17 illustrations. 1936. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 10; bound, RM. 11.50.

THE object of this small volume is to indicate, for the benefit of the general practitioner and the young surgeon, the methods which are available for producing anaesthesia, and to describe in some detail those which have been found most useful in the practice of a large city hospital in Berlin. In addition, the diagnostic and therapeutic uses of injection of local anaesthetics and of alcohol are considered briefly. Throughout the work stress is laid on the psychological state of the patient before and during operative procedures.

The first portion of the work enumerates methods suitable for minor and for major surgery, for accidents, and for military surgery. In the second portion, selected procedures are described in greater detail. A number of excellent diagrams clarify the pages devoted to injection methods. Inhalation, intravenous, spinal, and rectal anaesthesia are considered in turn, and the indications for the use of each type are clearly and fully discussed. For certain cases complicated and combined methods are described.

The selection of methods is the fruit of wide experience, and as such merits attention. The directions are short and clear, and the book achieves its purpose of presenting a succinct account of tried and proved procedures.

Elements of Orthopaedic Surgery. By N. ROSS SMITH, M.B., Ch.M. (Sydney), F.R.C.S. (Eng.), Orthopaedic Surgeon, Cornelia Hospital, Poole. With a Foreword by R. C. Elmslie, O.B.E., M.S., F.R.C.S. Lr. 8vo. Pp. 246 + xi, with 99 illustrations. 1936. Bristol: John Wright & Sons. Ltd. 10s. 6d. net.

IN this small manual the author has succeeded in presenting a concise and practical account of the elements of orthopaedic surgery. The common conditions treated by orthopaedic surgeons are described, and there are three appendices dealing with physiotherapy, splints, and plaster-of-Paris technique. The section on fractures is perhaps not as precise as the rest of the book. Thus, the statement that fractures of the scaphoid require only six weeks' immobilization in plaster for bony union to occur is misleading.

The work is clearly written and well illustrated, and should fulfil most of the requirements of nurses, masseuses, and others engaged in orthopaedic work who do not need the resource of the larger text-books.

Illustrations of Regional Anatomy. By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. Fcap 4to. Section VI, Upper Limb, 42 Plates, 7s. 6d. Section VII, Lower Limb, 52 Plates, 10s. 1936. Edinburgh: E. & S. Livingstone.

PROFESSOR E. B. Jamieson has, in response to numerous requests, added Sections VI and VII to his loose-leafed *Illustrations of Regional Anatomy*. These are very beautiful, most

helpful, and, as far as one can judge, quite accurate. It may be true, as the artist indicates, that the arm and leg do not lend themselves so fully to this form of representation as the trunk. We are glad to see that he uses the nomenclature provided by the Anatomical Society in 1933.

These plates will be particularly useful to two types of person whose interest in anatomy is vastly different: the student who has little visual sense; and the teacher of anatomy who is a poor draftsman, for the plates can be taken out of their covers and displayed in an epidiascope.

Fasciæ of the Human Body and their Relations to the Organs they Envelop. By EDWARD SINGER, M.D., Department of Anatomy, College of Physicians and Surgeons, Columbia University. 10½ × 7½. Pp. ix + 105, with 24 illustrations. 1935. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 13s. 6d. net.

THE author deplores the neglect by anatomical teachers of the fascial planes of the body, which he maintains are not only of anatomical interest but of considerable surgical importance. His object in writing this book is to counteract this tendency and necessarily perhaps to over-emphasize the definition of certain connective-tissue planes. Among most European anatomists there has been an increasing tendency during the past thirty years to regard these fascial layers as artificial—that is, as being largely produced by the ingenuity of the dissector—and of less importance than anatomists of the past century considered them. One cannot help admiring the crusader who is willing to back his opinions with such an interesting book as the author has produced. If he leaves one unconvinced he is certainly in no way to blame. One cannot help hoping that this detailed description is unnecessary, for otherwise he adds to the burden of medical students whose backs are already overweighted with anatomical detail. The letterpress is adorned by some first-rate illustrations; and the book is well produced and printed on high-class paper.

Disability Evaluation: Principles of Treatment of Compensable Injuries. By EARL D. MCBRIDE, B.S., M.D., F.A.C.S., Assistant Professor in Orthopedic Surgery, University of Oklahoma. Large 8vo. Pp. 623 + xvi, with 374 illustrations. 1936. London and Philadelphia: J. B. Lippincott Co. 38s. net.

THIS is an ambitious attempt to cover a very difficult subject. In the first chapter the legal aspects of compensation are considered and the position and function of the medical aspect defined. This is followed by a brief statement of the details of workmen's compensation laws in the various provinces of Canada and in each of the United States, for there are quite considerable variations.

The second chapter deals with the methods of evaluation of disability—a notoriously difficult subject. Detailed methods of trying to measure disability arithmetically are given, but it may be doubted whether any of these can really prove successful. The most useful part of this section consists of the diagrams illustrating such things as limited movement, delayed action, awkwardness, weakness, etc., in relation to work. These are graphic, and point the moral better than any tables of statistics. The rest of the book is devoted to methods of examination and then to a consideration of disabilities of regions. Here the author is handicapped by the evident fact that he has attempted to write for more than one class of reader. He has not written simply for the orthopædic or even surgical specialist, nor apparently purely for the medical profession. As a result the consideration given is sometimes too great, sometimes too little; this particularly applies to methods of treatment. Nevertheless he has attained a considerable success in formulating guiding principles upon the estimation of disability, a success in no small part due to the graphic illustrations. The book should prove of considerable value to those who are concerned with the workmen's compensation laws, more perhaps in America than in England, because in England the criterion is the ability of the workman to carry on his work, and the degree or percentage of disability does not concern the medical referee.

St. Bartholomew's Hospital Reports. Edited by W. G. BALL, G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, H. H. WOOLLARD, R. C. ELMSLIE, J. P. ROSS, and J. MAXWELL. Vol. LXIX. Demy 8vo. Pp. 413 + xxvi. Illustrated. 1936. London: John Murray. 21s. net.

As we have before observed, the *St. Bartholomew's Hospital Reports* are notable for the high standard of the notices relating to the passing of the members of their staff and school.

The present volume contains sympathetic articles on Holmes Spicer, Sir Archibald Garrod, and Sir Wilmot Herringham, who, in their own spheres, were distinguished and fully upheld the reputation of their old school. This volume also recalls Dr. R. G. Canti, who combined in a most unusual degree imagination and flair for research with an extraordinary knowledge of optics, photography, and electricity. As a mechanical genius he must have had few peers and no superiors. It is difficult at so close a view-point properly to assess the value of his contributions to medicine. One most notable side-line was his perfection of the technique for transfusion, and his assistance to the London Blood Transfusion Service, to which he was a scientific adviser, was invaluable. His work in the production of films of tissue growth is known throughout the world. Yet it was only a part of his main object, which was the cancer problem. The writer enjoyed frequent contact with Canti as a fellow examiner, and no one who met him in these circumstances could fail to appreciate his high mental ability.

It is seemly that an account of the new Medical College at Bart's should be written by the Dean, who is largely responsible for the success of the undertaking in all its aspects. The most notable fact of this transaction is the wonderful effort in financial assistance made by old Bart's men.

The clinical and pathological papers which constitute the remainder of the volume obviously contain the results of careful study, and will be of help to those interested in the particular condition; for the ordinary reader they may be regarded as difficult of digestion and of a type which one puts aside for future reading. Hospital Reports provide an excellent medium for the publication of such articles, which are really works of reference to which workers in their particular fields should have ready access.

St. Thomas's Hospital Reports. Edited by Prof. O. L. V. S. DE WESSELOW and C. MAX PAGE, assisted by N. R. BARRETT, J. ST. C. ELKINGTON, and A. J. WRIGLEY. Second Series, Vol. I. Royal 8vo. Pp. 199, with illustrations. 1936. London: St. Thomas's Hospital. 10s. (7s. 6d. to subscribers).

WE welcome the first volume of the new series of *St. Thomas's Hospital Reports*, and the more gladly because the previous series had fallen somewhat behindhand. Sir Thomas Smith once said of the *St. Bartholomew's Hospital Reports* that he looked upon them as his private Mausoleum, for he used to bury many good papers in them and they never saw the light again.

The editors of the present volume are Professor O. L. V. S. de Wesselow and Mr. C. Max Page, assisted by Mr. N. R. Barrett, Dr. J. St. C. Elkington, and Dr. A. J. Wrigley. Mr. Max Page writes a short account of previous issues of the *Reports*, from which it is clear that this is the third and not the second series. The first was edited by Mr. Flint South from November, 1835; the second began in 1870. The present volume contains a series of interesting articles by various members of the staff. Professor de Wesselow and Dr. W. J. Griffiths ask, Has diabetes a multiple origin? Dr. Card contributes a clinical study of sixty-seven cases of diabetic coma. Dr. Wrigley writes on the physiology of menstruation, with especial reference to endocrine therapy, Mr. Lowndes on modern methods in the diagnosis of plumbism. Professor Dudgeon has a well-illustrated article on the demonstration of particles of malignant growth in the sputum. Professor McSwiney writes of the involuntary nervous system, Mr. Barrett and Mr. Battle on decompression of the intestine by naso-duodenal suction, and Dr. Elkington on metastatic brain tumours. The care of the premature infant, and experiences of the Friedman test for pregnancy, are considered by Dr. Doayne Bell and by Dr. Bamforth.

The surgical side is well represented by Mr. Robinson, Mr. Mimpriss, Mr. Moseley, and Mr. Lindahl, who write respectively on prostatic obstruction and transurethral resection; the problem of abdominal pain of undiagnosed origin; fracture of the carpal scaphoid; the prognosis of inoperable carcinoma of the rectum; and progressive post-operative gangrene of the skin. From the special departments come the treatment of pituitary basophilism by roentgen therapy by Dr. Tchaperoff, the treatment of adolescent coxa vara by Mr. Forlong, and the operative treatment of Ménière's syndrome by Mr. W. A. Mill.

The illustrations in black and white and in colour are well rendered and well chosen. Altogether a most satisfactory opening volume to what should prove a long and valuable series.

Livre Jubilaire. Offert au Docteur ALBIN LAMBOTTE par ses Amis et ses Élèves. Edited by Dr. JEAN VERBRUGGE. Pp. 578, with several hundred figures. Bruxelles: Vromant & Co. Belgian fr. 150.

To very few men has it been given to have the greatness of their work recognized during their lifetime and to have this recognition in the form of a great international "Jubilee Volume".

Albin Lambotte was born on July 3, 1866, and the present volume is a celebration of his 70th birthday. His name will always live as one of the greatest and most scientific exponents of the direct operative fixation of fractures, or 'osteosynthesis' as he himself called it.

His father was Professor of Geology and Metallurgy at Namur, his elder brother a surgeon of distinction. At Antwerp all his surgical work was done. Like almost all great specialists Lambotte began as a general surgeon. The surgery of cholera and of diphtheria first claimed his attention, and between 1890 and 1900 he established a great reputation as an abdominal surgeon, and also by his work on the brain and spinal cord. He advocated and practised surgical intervention for cerebral hæmorrhage, opening the skull and removing the clot.

It was from 1900 to 1910 that he first devoted himself to the operative treatment of fractures, writing his well-known book on the subject in the latter year. At first he was greatly handicapped by the lack of suitable instruments; and he overcame this by making the instruments himself, whilst later he collaborated with Colin of Paris.

The esteem of the whole surgical world and the devotion of his friends and pupils is reflected in all the pages of this present volume. It is a most fascinating series of papers to all who have followed the history of the surgery of bones, from the day when Lister made such surgery safe, to the present day, when most surgeons consider that in spite of the triumphs of the open methods, conservative principles represent the line of greatest safety and most rapid functional recovery.

The contributors include French (16), Belgian (9), North American (6), South American (3), English (2), Austrian (2), Swedish (2), Spanish (3), Dutch (1), German (1), Russian (1), Romanian (1), Italian (1), Danish (1)—49 altogether.

It is natural that with Lambotte's life work in their minds a large number of the contributors should devote themselves to various methods of operative 'osteosynthesis', particularly as these methods lend themselves so well to illustration. It is impossible to describe or even catalogue the majority of these articles, but some of the outstanding may be mentioned. Leriche writes a fascinating article on the general subject of 'Bone Surgery'; Svante Orell of Styrso on the transplantation of 'Os Novum'; Jean Verhoögen and Van der Beken (Brussels) on 194 consecutive cases of 'Osteosynthesis'; Jean Verbrugge (Brussels) on the value of magnesium in the surgical treatment of fractures.

THE BRITISH JOURNAL OF SURGERY, which was founded when Lambotte's fame was at its zenith, is glad of this opportunity of joining in the world-wide tribute to his genius.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Irish Free State Hospital Year Book and Medical Directory, 1937. Demy 4to. Pp. 224. Dublin: O'Neill Publications Ltd. 10s. 6d. net.

Recent Advances in Orthopaedic Surgery. By B. H. BURNS, B.A., B.Ch., F.R.C.S., Orthopaedic Surgeon to St. George's Hospital, etc., and V. H. ELLIS, M.A., B.Ch., F.R.C.S., Orthopaedic Surgeon to St. Mary's Hospital, etc. Large post 8vo. Pp. 296 + viii, with 108 illustrations. 1937. London: J. & A. Churchill Ltd. 15s. net.

Recent Advances in Anaesthesia and Analgesia. By C. LANGTON HEWER, M.B., B.S. (Lond.), D.A. (R.C.P. & S. Eng.), Anaesthetist and Demonstrator of Anaesthetics, St. Bartholomew's Hospital, etc. Second edition. Large post 8vo. Pp. 284 + x, with 113 illustrations. 1937. London: J. & A. Churchill Ltd. 15s. net.

Hautdesinfektions-probleme. By JØRGEN ERNST. 6½ × 10 in. Pp. 174. 1937. Copenhagen: Levin & Munksgaard. Kr. 10.

Surgical Anatomy. By GRANT MASSIE, M.B., M.S.(Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Guy's Hospital; Lecturer in Operative Surgery, Guy's Hospital Medical School. Third edition. 9½ × 6½ in. Pp. 468 + x, with 153 illustrations, many in colour. 1937. London: J. & A. Churchill. 18s. net.

Scalpel and Sword. By SIR JAMES ELLIOTT. Demy 8vo. Pp. 215 + viii. 1937. London and Sydney: Angus & Robertson Ltd. 7s. 6d.

Vorträge aus der praktischen Chirurgie. Edited by ERICH LEXER. Heft 12. Diabetes und Chirurgie. By Prof. Dr. A. W. FISCHER, Director of the Surgical Clinic, University of Giessen. Large royal 8vo. Pp. 34. 1937. Stuttgart: Ferdinand Enke. RM. 2.

Traité de Chirurgie orthopédique. Published under the direction of Professors L. OMBRÉDANNE and P. MATHIEU; Editorial Secretaries, M. LANGE, G. HUC, and PADOVANI. In 5 volumes. Large royal 8vo. Vol. I. Physiology, Pathology, and General Treatment. Pp. 902, with 433 illustrations. 1937. Paris: Masson et Cie. Paper covers, Fr. 270; bound, Fr. 300.

Diathermy. By ELKIN P. CUMBERBATCH, M.A., B.M. (Oxon.), D.M.R.E. (Camb.), F.R.C.P., Medical Officer in charge of Electrical Department and Lecturer on Medical Electricity, St. Bartholomew's Hospital. With nine collaborators. Third Edition. Demy 8vo. Pp. 576 + xvi, with 168 illustrations. 1937. London: William Heinemann (Medical Books) Ltd. 21s. net.

Operative Surgery. By J. SHELTON HORSLEY, M.D., LL.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va., and ISAAC A. BIGGER, M.D., Professor of Surgery, Medical College of Virginia, Surgeon-in-Chief, Medical College of Virginia Hospitals, Richmond, Va. With contributions by C. C. COLEMAN, M.D., F.A.C.S., JOHN S. HORSLEY, jun., M.D., AUSTIN I. DODSON, M.D., F.A.C.S., and DONALD M. FAULKNER, M.D. Fourth edition. Large royal 8vo. In two volumes. Pp. 1387 + xxviii, with 1259 illustrations. 1937. London: Henry Kimpton. 63s. net.

Plastic Surgery of the Nose. By J. EASTMAN SHEEHAN, New York. Second edition, entirely rewritten. 10½ × 7 in. Pp. 186 + xx, with 131 illustrations in the text, including 7 in colour, and 14 plates consisting of 104 photographs. 1937. London: Humphrey Milford, Oxford University Press. 38s. net.

Fratturati in Pace e in Guerra. By V. PUTTI. Cr. 8vo. Pp. 111, with 5 illustrations. 1936. Bologna: Licinio Cappelli.

- Medizinische Praxis.** Band XXIII. Das Kropfproblem. By Dr. EUGEN BIRCHER, Principal Surgeon, Kantonspitals, Aarau, Switzerland. Large 8vo. Pp. 143 + xii, with 41 illustrations. 1937. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 12; bound, RM. 13.
- Lomboartrite e Sciatica Vertebrale.** By V. PUTTI. Large royal 8vo. Pp. 218, with 144 illustrations, some coloured. 1936. Bologna: Licinio Cappelli.
- Verzögerte Knochenbruchheilung und Pseudarthrosenbildung, ihre Ursachen und Behandlung.** By Prof. Dr. GEORG BRANDT, Director of the Surgical Clinic, Municipal Hospital, Mainz. 10½ × 6½ in. Pp. 175, with 223 illustrations. 1937. Leipzig: Georg Thieme. RM. 13.50; bound, RM. 14.80.
- Preoperative and Postoperative Treatment.** By ROBERT L. MASON, A.B., M.D., F.A.C.S., Assistant in Surgery at the Massachusetts General Hospital. Royal 8vo. Pp. 495, with 123 illustrations. 1937. Philadelphia and London: W. B. Saunders Co. 25s. net.
- Die Hirnkammerformen bei Hirntumoren.** By Dr. OTTO DYES, Dozent für Röntgenheilkunde, Würzburg. Vol. 52 of *Fortschritte auf dem Gebiete der Röntgenstrahlen*. 12 × 8½ in. Pp. 79, with 105 illustrations. 1937. Leipzig: Georg Thieme. RM. 16; bound, RM. 18.

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